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VOL. XXVII.

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HYSTERIA. In considering the physiology of man, or of any other class of animals, physiologists are liable to suppose that individuals may be looked upon as practically alike, though their attention must often be drawn to the fact that they react somewhat differently under observation. To the physician one of the most striking things is the difference between individuals, and this difference is one of the important factors in disease. We see this strikingly illustrated in families where, though the several members are much like those of other healthy families, in the course of their lives they show that they are not the same. They are born different; so that we may see, for instance, the arteries in one family slowly but surely degenerate, while those of another family retain their healthy peculiarities to an advanced age. In other families the kidneys degenerate early, or the gastro-intestinal canal shows itself incapable of carrying on the digestive processes in the steady, unerring way that one observes in others. These differences are, no doubt, due to the original laying down of the several systems and to the inherited conditions of their cellular constituents. There is no part which illustrates these observations better than the nervous system. One portion of a liver or a kidney is much like another, and the whole organ consists of an agglomeration of similar lobes and lobules, which are very like those in lower animals. But the nervous system of man, and to a less extent that of animals, consists of a great variety of sub-divisions and systems which are not alike. And in the ascending scale of living beings new and higher parts are added. This increasing complexity of the nervous system reaches its maximum in man. The stability of any portion of the nervous system, that is to say, its power of resisting causes which derange function, is, other things equal, proportional to the length of time during which it appears to have existed as a part of the nervous system. In man the cerebral cortex is the part which has been most recently developed, and one might expect that it would frequently show signs of functional disturbance. But even amongst the apparently healthy there are the greatest differences as to the same spasm; it is, in fact, a case of ideational automatism, and of cortical origin. If the view that the cerebral cortex is the seat of functional derangement in hysteria is correct it accounts for the great variety of symptoms met with in the disease. For not only are general and special sensation, voluntary movements, speech, emotion, &c., arranged for in the cortex, but the action of the heart, respiration and other processes which may be deranged in hysteria, are at least influenced by cortical action.

Wherein, then, lies the difference between the normally acting brain and the hysterical? Many hysterical people are bright, intellectual and attractive. But their brain action is characterised by three peculiarities at least: (1) Explosiveness, if I may say so; (2) want of control; (3) proneness to exhaustion. So that two sets of phenomena occur: one a too active and uncontrolled nerve discharge, the other paralysis of various kinds, sensory and motor.

Emotion, for example, in a healthy person initiates muscular movements of various kinds, expressions of joy or sorrow, as the case may be, controlled, however, within appropriate limits; while the same cause of emotion may, in the hysterical subject, reverberate impetuously and unrestrainedly through the motor centres, mingling the natural expressions of joy and sorrow confusedly together, and producing irregular movements of the limbs, or even general convulsions.

In other cases emotion inhibits nerve action and produces various forms of sensory and motor paralysis. Or the latter may be due, not to inhibition, but to exhaustion of cells of the cerebral cortex.

The exciting or restraining influence may be central, such as an emotion or an idea, but it may also be peripheral, as is shown by the various points, whether superficially or deeply situated, pressure upon which either gives rise to a hysterical fit or stops one which has already commenced.

The explanation of muscular spasm and of sensory and motor paralyses in hysteria is rendered very difficult by the fact that parts of the brain can certainly inhibit nerve

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thermoanæsthesia or electroanæsthesia, or alterations in muscle sense, but tactile sensation is the last to go, and never disappears alone. The explanation of this is uncertain. In disease of the distant peripherally distributed sensory nerve-fibres it is much more easily affected, for if the receiving terminals are paralysed no external impressions can enter upon the upward path to the cerebral centres, however healthy the latter.

In the cortex itself, if one may be allowed to speak of the "field of sensation" as one does of the field of vision, the area of tactile sensation may perhaps be compared with the yellow spot in the retina, and both may be looked upon as anatomically constructed to be the parts most sensitive to impressions, each in its own field. And just as there may be a dissociation of sensations, certain forms—such as those of pain and temperature—disappearing while tactile sensation remains, so in the eye vision may be retained, while colour vision may be altered, violet and green disappearing first, and blue, yellow and red later. Such points as these, however, must be left to the physiologists to explain.

But the visual changes mentioned are not the only ones which occur in hysteria, nor, indeed, are they the most important. The most characteristic alteration is the concentric contraction of the fields of vision. So marked may this be that the fields may be reduced to very small circles around the yellow spots. In one sense the change is symmetrical, that is to say, it occurs in both eyes; but in another sense it is generally not so. If a hysterical patient has, for instance, hemianæsthesia of the left side of the body, this includes vision and hearing too. But the change in the fields of vision is not like that which occurs in hemianæsthesia from gross lesions; it does not consist of hemianopsia, but of a concentric retraction of the field around the yellow spot. It is doubtful whether hemianopsia ever occurs in hysteria; I have never seen it myself. But whether it occurs or not, concentric retraction of the fields is the change which occurs almost invariably. On the hemianæsthetic side, however, the contraction is far greater than on the other.

Now, I would ask the physiologists how this state of things can be explained. It hardly tallies with the view that the homonymous lateral halves of the retinæ are in relation with a half vision centre in the corresponding occipital lobe, and that each hemisphere has no more intimate relation with the retina on the opposite side than with that on its own side. The dynamic changes which occur in the brain in hysteria are no doubt bilateral, and do not, as a rule, confine themselves to one hemisphere only, though they affect one more than the other. But if it be assumed that the retina, on each side, while it is in connection through the occipital half vision centres with both hemispheres has also a more direct connection with the hemisphere of the opposite side, this would explain how the field of vision is most contracted on the side of the body on which the other sensations are paralysed. A different explanation of the phenomenon is, however, possible. The contraction of the fields of vision may be a peripheral change and not a cortical one. The retina is well known to be less and less sensitive as one passes from the yellow spot to the periphery; and consequently if there are changes which act in a paralysing manner upon the distribution of the optic nerves, the effect will naturally show itself most where the function of the retina is normally least vigorous; while the function of the most sensitive parts about the yellow spot will be the last to be extinguished. But there is no good reason for looking upon hysteria as a disease of peripheral nerves; it is a disease principally of nerve centres. We must, however, remember that the retina in its origin is really a process from the brain, and not strictly speaking a peripheral nerve.

The occurrence of anæsthesia in hysteria is not confined to the skin and special senses, the mucous membranes and deeper tissues may also be affected. Anæsthesia of the pharynx, for instance, is of great importance in the diagnosis of the disease; and Pitres asserts that patients often tolerate, and even enjoy, the hardest pressure that can be applied to the epigastric region. Even the periosteum and bones may be analgesic. Sometimes, on the contrary, marked on the left side, absence of plantar and pharyngeal reflex, marked analgesia of the left leg and the left half of the body, diminishing from below upwards and sparing the face.

But it is probable that the functional inertia of nerve-cells is sometimes present in the cord of such cases as well as in the brain, for some of them have flaccid paralysis of limbs and absent plantar reflex, although another explanation is possible, viz., that the pyramidal tract is over-acting and so inhibiting the reflexes of the cord. It may be objected that one cannot explain both hyperactivity of the cord cells and inertia as well by supposing that in the same disease the pyramidal strands are sometimes over and sometimes under-acting. But this very uncertainty in the action of nerve centres, which is sometimes exaggerated and sometimes depressed, even in the same case, is one of the great characteristics of hysteria. Hence arise the various forms of hyperæsthesia and spasm on the one hand, and anæsthesia and paralysis on the other. In a healthy person, with normal, stable nervous system, there is an equable, steady current of nerve force, passing from centre to centre, resulting in the production of a state of equilibrium and healthy, mutual control. In hysteria there seems to be diminished pressure, if I may say so, so that energy ebbs and flows, and centres sometimes exhibit explosions of activity, and sometimes become almost inert. The activity of the latter may, however, suddenly revive and a paralysis of movement or sensation disappears, owing to various causes, such as an access of anger, joy, fright, &c. The sudden transition from inactivity to activity shows that there can be no fundamental change in the cells, but only a dynamic one.

How do such redistributions of nerve energy arise? Can it be that there are certain cells amongst the millions which exist in the layers of the cortex the function of which is to generate nerve force for distribution to the various centres? It is certain that the exhaustion of some regions entails the exhaustion of others which have not been in activity at the same time; intellectual work, for instance, causes brain fatigue which renders physical work difficult, and *vice versa*. This suggests that there is either a common source of energy on which the various regions of the cortex draw when in active work, or else that nerve force can flow from one to another, or perhaps, as others suggest, that the fatigue products of centres in action act detrimentally on the rest. Whatever be the explanation, in hysteria some centres may retain their usual activity, while others are inactive, and produce various forms of paralysis, while the extent and intensity of the loss of function may vary almost from day to day.

In the Gulstonian Lectures, which I delivered in 1886, I wrote as follows: "Some think the centres are starved by contraction of their nutrient blood-vessels. It seems to me more likely that the nerve centres are exhausted by their own action. Thus, sudden emotional excitement may bring on hysterical phenomena indicating nerve exhaustion, such as hemianæsthesia, paralysis, or spasm. It is important to remember that the exhaustion of one area seems to affect the whole system. This may indicate that the various differentiated centres draw force from some common supply. Considering the enormous number of cells which are found in the cerebral gray matter, many without evident connections, is it going too far to suppose that a large number of these are factories for the production of nervous energy which is necessary for the continuous and regular action of the highest centres, and which flows in increasing quantities towards those centres which are from time to time brought into action? Constant supply would mean constant power in all centres, and, as the latter are bound together so as to produce a physiological equilibrium, constant power would involve constant control of one by the other.

"When the store of constantly produced force (represented, of course, by structural peculiarities) is by inheritance small, or when it has been exhausted by unnatural calls upon it, there would be insufficient to supply all the centres, and those to which the nerve paths are most freely open by habit would command it, while others would be starved, and loss of equilibrium, or the natural control of one centre by the healthy action of others, would be the inevitable result. If we do not suppose any such reservoirs of nerve energy, how can we explain the exhaustion of all by the excessive action of some centres? Why is a person who has had excessive mental work less able for the time to do active bodily work? And why when bodily fatigued are we less able to do intellectual work?"

In BRAIN, Part II., 1903, Dr. McDougall has a paper on "The Nature of Inhibitory Processes within the Nervous System," in which he develops a similar idea. Here are some quotations from his article: "Inhibitory effects in the nervous system result from a process of competition between the inhibitory and the inhibited tract, the former draining to itself a part of the energy liberated in the latter." "Inhibition appears always as the negative or complementary result of a process of increased excitation in some other part." "The mind has only a limited quantity of energy, which will not suffice for the simultaneous maintenance of two mental processes." "Inhibition is due to competition among nerve paths for the living energy present at the moment in the nervous system." "The relation of reciprocal inhibition obtaining between all the different organised neural systems, constituting paths of forward conduction in the higher brain levels, is due, I suggest, to the fact that they all drain one common store of neurin contained and constantly generated in the interconnected mass of afferent neurones, and seeking constantly to escape by the paths of least resistance into motor neurones, and so into the muscles."

Amongst the hysterical manifestations which occur in such variety are some which suggest abnormal conditions of centres in the medulla oblongata or in the sympathetic system.

Well-marked cases occur in which respiration and circulation are affected. The movements of respiration may be slowed or accelerated, and so may the heart's pulsations. The following is a remarkable instance:—

A girl, age 21, began in May, 1886, without evident cause, to have a feeling of fainting, or "catching in her breathing," when in hysteria and allied conditions. But whether we clearly understand the abnormal processes going on in the nervous system in hysteria or not we have to treat the victims of it to the best of our ability.

If the disease be looked upon as a congenital condition we cannot hope to cure it, that would involve rebuilding the nervous system *ab initio*. But, as has been already remarked, latent stigmata may be the only evidences of the condition, and further manifestations which seriously interfere with the uses and pleasures of life may be absent. Methods of prevention and those directed to cure must follow similar lines. Broadly speaking, the well-known laws of health must be observed, and a quiet, pleasurable, contented life must be secured if possible. When serious symptoms of the disease have already manifested themselves the recovery of losses in general nutrition may be greatly assisted by massage, if accompanied by rest. But massage may positively aggravate the disease if it is employed while the patient pursues the same exhausting life which has led to the breakdown. It is then only adding one more cause of exhaustion. In most instances rest in bed for some weeks — six to eight -- is required. The upsetting effects of emotions, whether of joy, or still more of sorrow, and the worries of every-day life, have a much greater effect upon hysterical people than upon the healthy, therefore to save them from these isolation is requisite; but unless great care is taken this may do the patients more harm than good. It is not uncommon to find that medical men, having correctly diagnosed a case as hysteria, and having also rightly recommended isolation, massage, electricity and good feeding, send for a nurse to take charge of the patient, without taking any serious thought whether the two will make suitable companions. And yet this is the most important point in the whole treatment. One sees the effects upon ordinary healthy people of constant association with an uncongenial companion; but under such conditions a person can at least be relieved of the companionship for longer or shorter periods of the day. If a hysterical person is made to live with a nurse she does not care about, or even dislikes, she cannot escape from her and the results will be disastrous. If, then, the medical man desires to restore his patient to health he must take the greatest pains in selecting the nurse who is to take charge of the case, and he must, if necessary, discharge one after the other till he gets the right one, if the two are not found to be happy together. And no nurse ought to resent being relieved of such a charge for such reasons; no one gets on smoothly with everyone they chance to meet. Looking upon hysteria as mainly an abnormal condition of the brain of a functional kind, one would expect to be able to influence it mainly through healthy ideas and pleasurable emotions; and just as ideas and impressions received from another case may lead to the abnormal movements called chorea, so ideas and impressions derived from the association with healthy-minded, congenial companions may lead back to healthy conditions of brain action. By far the most important of all considerations in the treatment of hysteria is, then, the selection of those who are to be with the patient, and most of all, of the nurse who is to take charge. But the masseuse is almost of equal importance. Medical men are inclined to think that if they supply a skilful masseuse that is all that is required. But consider: the masseuse spends generally two or three hours a day with the patient, and her influence on the patient's mind may have as great an effect as her massage in favouring (or the reverse) a return to health. Personally I am in the habit of considering, as far as possible, the character of every one who comes into contact with hysterical patients, knowing full well that personal influence plays the most important part in their recovery. This is the natural and healthy form of "suggestion," and it is free from the dangers connected with that which is more technically called "suggestion."

As regards drugs one may say that they play a subordinate *rôle* in the treatment of the disease. Tonics, of course, help to restore the physical condition if it has suffered, but strychnine, one of the most active nerve tonics we possess, generally produces discomfort. Bromides are almost universally employed; and looking at the view we have enunciated as regards the pathology of the disease one can well see how they can, by depressing the energy of the cortical centres, diminish the hyperæsthesia and irregular nerve discharges which occur. But the same effect will be produced upon those centres which are already unable to function normally, and will increase the sensory and motor paralyses, and diminish the controlling and energising nerve discharges which tend to a re-establishment of the lost equilibrium. Bromides, therefore, ought to be used in great moderation, as drugs which may relieve the discomforts of hysteria, but have a tendency in the long run to retard recovery.

Another disease of the nervous system of great importance is NEURASTHENIA. In some ways it resembles hysteria, and yet it is very different. Inheritance probably plays an important part in both ; that is to say, not direct inheritance of the disease itself, but rather of a central nervous system which lacks certain characters which a perfectly sound one possesses. As we are in ignorance as to the actual pathology of either one or the other condition it is very difficult to describe what those characters are. In hysteria there is an absence of that steady, equable, well-balanced nerve action which is seen in healthy people. The consequences are explosive discharges on the one hand and sensory and motor paralyses on the other. In neurasthenia no such symptoms attain prominence ; but a condition of simple exhaustion of nerve energy is the main characteristic.

Inheritance presents itself in the form of a tendency to the production of such exhaustion ; an absence of staying power ; an incapability of enduring unscathed the amount of stress and strain which a healthy nervous system can endure. Hence it is more common in men than in women ; as the mental and physical strains which men have to bear are greater than those which befall the opposite sex. And hence, too, the proneness of the condition to occur in hysterical subjects, who possess a nervous system which is easily exhausted. Neurasthenia is supposed to be much more common than it used to be, and, looking upon it as an exhausted condition of the highest nerve centres from strain, the conditions of modern life account for this. Its onset is generally gradual, and slowly produced by life's worries, anxieties, and overwork. But it may occur suddenly after physical injuries; though even in this case weeks may often elapse before it declares itself. In relation to this fact it is of interest to note that Obersteiner says it has been shown that injuries, such as blows on the head, may give rise to degeneration of nerve-fibres which extends a long way into the nervous system.

A sudden emotion may likewise produce profound neurasthenia. A patient of mine suffered from an absolutely incapacitating attack of the disease owing to hard work, exposure to sun in India, and other unfavourable conditions. He recovered very slowly, after a year of treatment, but so completely that he went through the South African campaign without difficulty and returned home well.

After an interval of six years from his former attack, during which he said he had been in better health than he had ever been, he returned to me physically incapable of going about and unable to do mental work, or even to read a book or a newspaper. He had stubborn insomnia, and great hyperæsthesia as regards sounds. He was, indeed, fit for nothing. This state of affairs had developed quite suddenly, owing to his being refused by a lady, whom he had known for a considerable time, and who had given him reason to believe that a proposal of marriage would certainly be accepted. The sudden emotional shock produced by the refusal resulted in a sudden and complete breakdown.

It has already been remarked that one is born hysterical, and in a similar sense it may be said that one is born neurasthenic. In both cases one is born with a nervous system which will show its defects under unfavourable circumstances. But, as has also been remarked, such unfavourable circumstances may never overtake the person; and so the weakness is never discovered. Neither fits, nor sensory and motor paralyses, nor contractions, occur as a rule, in neurasthenia. The most striking phenomena are mental and physical weakness; a condition in which fatigue easily follows muscular or mental exertion even of a very moderate kind. The patient cannot, for example, apply himself to his business as he used to do; he cannot concentrate his attention; his memory is defective. Nor can he, owing to inertia, indulge in the recreations and exercises which before gave him pleasure. One of the most prominent symptoms is insomnia, which is not only a result of his disease, but also aggravates it. Restlessness, fretfulness, irritability of temper, hypersensitiveness, visual and auditory, discomforts about the head and upper part of the neck, especially posteriorly, may all be sources of great trouble. Things which never caused worry before do so now. This hypersensitiveness on the mental side has its counterpart on the physical. Pains and discomforts may arise in all parts of the body, suggesting the presence of disease and causing anxiety to the patient. The various organs of the body do not ordinarily in healthy conditions force themselves into notice. But the neurasthenic patient may be painfully conscious of his natural need of breathing: of the action of his heart; of the presence of food in his stomach; of the presence of small quantities of urine in the bladder or of fæces in the large bowel, and, indeed, of almost everything which is going on in his body, which the healthy man does not perceive, and these abnormal sensations are not only uncomfortable but are also disquieting, as suggestions of all kinds of diseases. He suffers from indigestion, flatulence, and a sinking in the epigastrium. A dread that something serious is going to happen may haunt him; and he may be troubled with agraphobia and claustrophia. The deep reflexes are exaggerated as a rule, and a very constant phenomenon is a feeble pulse of very low tension.

Neurasthenia is the result in most cases of prolonged adverse conditions, which make constant calls on the highest nerve-centres without due intervals of rest and relaxation being allowed for recuperation. Constant worry and strain of one kind or another are almost always the antecedents, though it is true that sudden emotion on trauma may have similar effects. But, as has been pointed out, even under the latter circumstances, the condition may not follow at once, but after an interval of a few weeks. The disease is due to sheer exhaustion of the highest nervous centres, and this exhaustion is pretty certainly accompanied by alteration in the nerve-cells—in fact, by degeneration. In hysteria this can hardly be the case; for, if it were, the sudden disappearance of motor and sensory paralyses could not occur.

The persistence of neurasthenia, and the length of time which it takes to disappear, also speak in favour of a degenerative change having occurred in the nerve-cells, at least of the cerebral cortex. Indeed, I believe that many patients never succeed in regaining the condition they enjoyed before they broke down, but remain incapable of doing the work they used to do. Even at their best they can no longer work with rapidity, or stand worry. They have to reduce themselves to a slow, steady pace, and then, and only then, can they accomplish much.

Recent investigations have shown that anatomical alterations occur in nerve-cells when acting in an ordinary way as well as when pushed to fatigue. van Gehuchten sums up the results of these investigations as follows:—

The cellular protoplasm during normal activity undergoes swelling, which produces an increase in volume of the body of the cell and diminution in the quantity of the chromophile substance.

The nucleus likewise augments in volume.

When pushed to fatigue there is a diminution in the protoplasm of the cell, and the nucleus diminishes also, and undergoes an alteration in shape and modifications of its chromatic substance.

Hodge found similar alterations in the nerve-cells of fatigued animals, such as the pigeon and the honey-bee. These disappeared after a night's rest. In neurasthenia, as a rule, the antecedent stress and fatigue have been prolonged, and there has been but little opportunity of recuperation owing to the accompanying insomnia. Hence on[illegible] would expect much more profound atrophy and other pat[illegible]logical changes in such cases.

As regards traumatic neurasthenia which follows [illegible] railway and other accidents, it can hardly be doubted that the nervous system, and especially the brain, is actually physically injured. In examining *post-mortem* cases which have succumbed to their injuries one often finds more or less widespread bruising of the surface of the brain. This must involve the neuraxons and dendrites of many neurones. Recent researches have proved that not only section but many injuries of the neuraxon involve not only peripheral changes in it but also alterations in the nerve-cell from which it emanates. van Gehuchten gives the following: Alteration in the chromophile substance—chromolysis; swelling of the cell body; displacement of the nucleus to the periphery.

The chromolysis commences about forty hours after the injury, and lasts fifteen to twenty days. Then occurs a re-formation of the chromophile substance and a return to normal in about 100 days. The cells, however, may atrophy and disappear, the whole process being more or less rapid and complete according to the severity of the injury.

As regards afferent nerve-fibres it has been shown, at least in the case of those of the spinal ganglia, that when they are injured changes occur in the ganglion cells of a similar kind, only more rapidly, and destruction of the cell follows. It is probable, therefore, that injury of the protoplasmic processes or dendrites in nerve centres would entail changes in the cells with which they are connected. In a severe concussion of the brain, therefore, it may be assumed that such changes occur in a considerable number of neurones; and as one neurone transmits stimuli to others, the function of many uninjured neurones would probably be altered by a diminution in their normal afferent stimuli. Barker, in his book entitled "The Nervous System and its Constituent Neurones," 1900, says: "The importance of normal adequate stimuli for the welfare of the neurones in health can no longer be questioned. As van Gehuchten [illegible]as emphasised, without stimuli there can be no life. For [illegible] maintenance of absolutely perfect function, the relation [illegible]imuli to the reparative nutritive power of the cell must [illegible]olutely and perfectly adjusted."

Sherrington has shown how division of the dorsal nerves of the cord produces a loss of tone in the cells of the anterior horn. In a delicate and complex organ like the brain it can easily be imagined, therefore, what changes in its function, as a whole, might be produced by such pathological alterations in certain territories, intimately connected as the neurones are with each other.

But it may be objected, how is it that many such cases get rapidly well after the action for damages which they have instituted has been settled, but not before?

The trial does not generally come on till a long time after the injury has been received, and during that period the damaged nervous system has been gradually returning to the normal, a process which experiment shows to be a lengthy one. But meanwhile other factors, such as mental depression with reference to the loss sustained by prolonged illness, anxiety about the future, including the question whether damages will be granted to the patient by the court, have been added, factors which of themselves might produce neurasthenia. When the trial is ended, and suitable damages have been obtained, the mental causes of the patient's neurasthenic condition are removed, and satisfaction and hope are free to exert their tonic influences on a nervous system which time and treatment have rendered capable of responding. To procure this result, it seems to me only natural that the railway company or other agency which has inflicted the original injuries should be made to pay, as they alone can administer the medicine which will cure the incapacity for which they are responsible.

To what extent spinal centres and the sympathetic system are involved in neurasthenia is as doubtful as in hysteria. The fatigue of body and mind which the victims of neurasthenia present can be quite accounted for by degenerative changes in the motor and other regions of th
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down and the patient become the victim of neurasthenia. Those who start life with a bad inheritance from the nerve side, and who have a tendency to develop this disease, may as regards quality of nerve-action be amongst the most brilliant members of our race; but it is in quantity, or staying power, that they are deficient.

Prolonged illnesses, and especially the specific fevers, may leave the patient in a neurasthenic condition; and of all of them perhaps influenza is the worst. Febrile diseases, due, as they generally are, to micro-organisms and their specific poisons, are well known to produce granular degeneration and other changes in the cells of the body; and it is highly probable that the neurasthenia they leave behind is due to such changes in the cells of the highest nerve centres.

Neurasthenia is one of the most seriously incapacitating conditions, and its long duration, even under the best treatment, makes it very trying both for the patient and his friends; and what makes it even worse is the view which the relations and others often take of the case. Seeing the patient without any tangible local disease, or notable derangement of functions, but full of complaints and professing incapacity to do many things for which they cannot see any reason, they label him as "hysterical," and think that he shirks work and duties, and even pleasures, in which he could quite well take his share. But the incapacity is very real and requires careful and prolonged treatment.

If, as is supposed, a real degeneration of nerve-cells has occurred in neurasthenia, the length of time which the disease lasts, the slowness of the progress towards recovery, and even the failure of the patient ever to regain the condition of nerve energy which he formerly possessed, are accounted for. The main factor in treatment must be rest, [illegible]ntal and physical; not idleness, however, except in [illegible]me cases. In the latter, where exhaustion is pro-[illegible] the patient ought to be put to bed and the nervous [illegible]ested. It is advisable, however, that the circulation [illegible]ondition of the muscular system should be main-[illegible]thy, active condition, and this massage, with the aid of electricity, can accomplish, without any effort on the part of the patient's nervous system. Isolation may, or may not, be advisable. In some cases mere talking with friends fatigues, and it is better for the patient to be alone, with nurses only to attend him, for a while. But isolation in neurasthenia is required for purposes of rest, and not, as in the case of hysteria, so much as a means of bringing fresh and healthy influences to bear upon the patient. Plenty of wholesome food, and sometimes a small quantity of alcoholic stimulants, should be given. Insomnia is a great trouble, and narcotics must, in many cases, be administered, in order to obtain a moderate amount of sleep. Chloral hydrate, chloralamide, trional, &c., may be given in sufficient quantities to produce a good night's rest two or three times a week, but not every night. After a few weeks of this treatment the patient may be allowed to get up for longer and longer periods, be taken out to drive, and then allowed to walk. But whatever he be allowed to do must not be enough to produce discomfort, which really means fatigue; both mental and physical work and pleasure must fall short of this. And, in my opinion, in all cases of neurasthenia this is the most important point in the management of the patient, namely, the prevention of his doing more than that which leaves him without fatigue, the amount he can perform will then slowly but gradually increase. It is easy to lay down this rule, but an exceedingly difficult thing to carry it out, owing to the long duration of treatment. A busy man, for instance, wants to go away for a few weeks and then return to full work. But this he cannot do. Indeed, it may be said that in bad cases of neurasthenia the patient must permanently alter the conditions of his life, reducing his hours of work, and increasing the time spent in recreation and exercise; because the very fact of his having broken down shows that he has done more than he individually is capable of doing with safety. Patients will not, however, easily admit that there are individual limitations of power and endurance, and take it for granted that what others can do they can. Drugs indirectly help the patient considerably, procuring sleep, aiding digestion, which is

A CASE OF "SUBACUTE COMBINED SCLEROSIS," WITH PROFOUND ANÆMIA.[1]

BY JAMES TAYLOR, M.A., M.D., F.R.C.P.

I FIRST saw the patient whose case I am about to relate, through the kindness of Dr. J. G. Fletcher, on July 10 of last year (1903). She was a woman of 63, and was sent to me on account of weakness of the legs and difficulty and unsteadiness in walking, which had commenced in December of the previous year, and had become gradually worse. There was nothing of significance in her previous health, except that for two or three years she had not felt well and her friends had remarked on her pallor and sallowness. Her illness began with a feeling of weakness in the legs. To this weakness was gradually superadded unsteadiness, and she was noticed to lift her feet quite unnecessarily high in walking. There had also been some wasting of the limbs and a feeling of weakness in the arms.

Tingling and a sensation of deadness in the legs had been a marked symptom ever since the commencement of her illness. There had also been some shooting pains in the legs, and she had had difficulty in passing water and occasionally incontinence of urine.

On examination at that time it was seen that her skin was of a pale yellowish tinge. Her hands seemed to be a little awkward, and there was distinct unsteadiness in standing and a high-stepping ataxic gait in walking. I could make out no sensory impairment, but she complained of shooting pains in the legs and of a sense of constriction round the waist. Both knee jerks were very active. I could find no ankle clonus, and I have, unfortunately, no note of the plantar reflex. She was extremely hopeful and sanguine, much more so than her condition seemed to warrant.

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[1] Read at the Harveian Society of London.

I regarded the case then as one of sclerosis associated with anæmia, and I anticipated that she would become worse. Her progress was steadily downwards, and on September 14, at Dr. Fletcher's request, she was admitted to the hospital.

On admission she was found to be much worse than when I had seen her, and I am indebted to Dr. Gordon Holmes for the very careful notes of the case during the time she was in hospital. She was quite unable to walk, she was much emaciated and weaker and her upper limbs were much wasted, and rigid and tremulous in the performance of fine movements. With the arms extended and the eyes closed there were athetoid movements present in the fingers, similar to those met with in tabes. The lower limbs were much wasted, very rigid, and subject to occasional severe spasms. There was much numbness and tingling in all the limbs, a girdle sensation at the waist, and dull aching pain in the lumbar region. There was definite impairment of sensibility to touch, pain and temperature all over the lower limbs. The upper limit was not definite. Stereognostic sense and sense of position were lost in all limbs. The knee jerks were active, there was slight ankle clonus, and the plantars were of the extensor type. She was quite unable to stand.

She gradually became much worse. On September 23 it was noted that her lower limbs were now flaccid and the knee jerk was not to be obtained. There was also complete incontinence of urine. Her muscles wasted, she became weaker and unable to feed herself, and her mind wandered at night. The anæsthesia had extended up to the second rib. The blood showed no qualitative change and there never were any retinal hæmorrhages present. She gradually became quite helpless; a bed sore developed; she usually wandered at night, but during the day her mind was wonderfully clear; she still showed the same hopeful and sanguine temperament which I had noted on first seeing her. She gradually sank, and died on September 27.

Of the *post-mortem* appearances I need only refer to the change in the nervous system, as the other organs disclosed nothing more than one would expect to find in the organs of a patient so emaciated and anæmic as she was. And I wish to acknowledge my great indebtedness to Dr. Farquhar Buzzard for the exceeding care he has given to this examination and for the excellent and full report with which he has furnished me. The brain showed no obvious abnormality. In the cord (figs. 1, 2, 3) there was extensive degeneration in the posterior columns, the only parts escaping being close to the gray matter. From the third lumbar segment downward

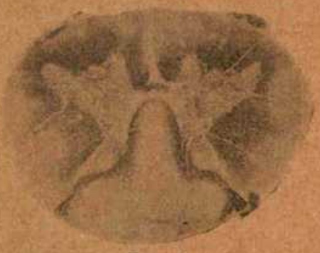

FIG. 1.

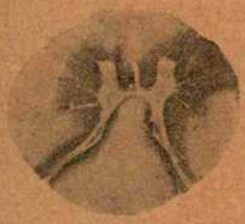

FIG. 2.

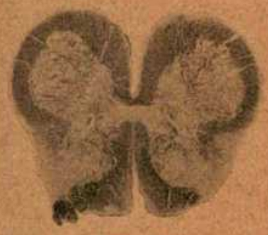

FIG. 3.

Sections in cervical, dorsal and lumbar regions of spinal cord.

the sclerosis rapidly diminished. In the first sacral was seen. The lateral columns were also sclerosed and here also the healthiest part was that next the gray matter; the crossed pyramidal, Gowers' tract and the direct cerebellar were all affected. The anterior columns showed less degeneration, except in the mid-dorsal where they were entirely degenerated, the only fibres escaping being those of the anterior limiting layer. Some degeneration was visible in the direct pyramidal tract as low as the fourth lumbar segment. The gray matter, so far as its myelinated fibres were concerned, did not seem to be altered. There was no degeneration in the posterior roots or in the anterior. The anterior horn cells were only slightly altered, apparently the result rather of malnutrition than of degeneration.

The cells in Clarke's column on the other hand had mostly undergone much change. They were nearly all shrunken, and many of them took no stain, being only represented by small masses of greenish pigment, retaining more or less the original shape of the cells. The cells of the posterior ganglia were also markedly pigmented and diffusely stained, rendering their details and outlines ill-defined.

The vessel walls did not appear pathologically altered, although they sometimes presented a hyaline appearance in sclerosed areas. The most noticeable condition was the small round-celled infiltration of the vessel walls, and of the surrounding lymph spaces. This was to be seen affecting many vessels in the posterior columns, and constituted a feature which is not common in cases of this kind. The membranes were unaltered, and the muscle fibre from the anterior tibial muscle showed no definite morbid change. The individual fibres were, perhaps, smaller than in a healthy subject.

In March, 1895, I read before the Medico-Chirurgical Society a paper on "Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anæmia." In this paper I described two cases in which the symptoms briefly were: gradual paraplegic weakness associated with impairment of the reflexes, with discolouration of the skin, in one case so severe as to suggest at one stage Addison's disease, with sensory impairment, great anæmia and emaciation, and finally death. In those two cases there were found to be extensive degenerative changes in the spinal cord of the nature chiefly of sclerosis of the white matter in the posterior and lateral columns, and also in the anterior. A case exactly similar both in its clinical and pathological conditions had been published a few weeks before in BRAIN, by the late Dr. H. Bowman, and as this patient had been under my care as an out-patient at Queen Square, and as I had frequently seen her when she was in the hospital under that of Dr. Hughlings Jackson, I had the opportunity of comparing her clinical progress with that of the patients whose cases I published, one of whom was in hospital at the same time. Further, I had made the *post-mortem* examination of Dr. Bowman's case, and had prepared the sections of the spinal cord which he and I had examined and compared with those from the cords of my patients. The clinical histories in all these cases were very similar. In Dr. Bowman's case there was a history that her illness began with a severe flooding, and when she came first to see me as an out-patient suffering from intense headaches, she was in a profoundly anæmic state, and her appearance was so cachectic as to suggest malignant disease. She was admitted to hospital, and under the influence of rest, good food, and arsenic and iron, she improved very much and went out almost well. About six months later she returned, and by this time paraplegia had developed, and she rapidly became worse. In one of my cases there was a history that four years previously, in Canada, he had had a similar condition of weakness of the legs and ataxy from which he had made a complete recovery. In regard to the changes in the spinal cord in Dr. Bowman's case and my own, these were practically identical.

In none of these cases could the blood condition or the condition of the organs after death be described as characteristic of pernicious anæmia; but the cord changes found were obviously identical with those described by Lichtheim in 1887, as occurring in certain cases under his observation of pernicious anæmia, and the clinical symptoms of paraplegia were similar. Minnich, his pupil, had also done some excellent work on this subject in the investigation of spinal cords from cases of pernicious anæmia in which paraplegic symptoms had not been present. In these he found scattered sclerotic foci, apparently the result of small hæmorrhages occurring in the cord, and also sclerosis in the posterior columns of varying intensity in different cords. He also found similar changes in the cords of patients who had suffered from other cachectic conditions, three cases of chronic jaundice, one of leukæmia, and one of tumour of the inferior vermis of the cerebellum. Lichtheim regarded the cord changes in his cases as the result of a toxic blood state, and Minnich's observations, while confirming such a view, suggested that some part of the sclerosis might be the result of hæmorrhagic foci in the cord, the hæmorrhages being similar to those occurring in profound anæmia, in the retinæ and elsewhere. It may be mentioned that in Dr. Bowman's case and in one of mine there had been retinal hæmorrhages observed before death.

During the last ten years numerous cases of a character similar to these have been published. Two important contributions came from Queen Square, continuing and supplementing the earlier observations of Dr. Bowman and myself, one by Dr. Risien Russell on three cases which he had observed, the other by the same writer associated with Dr. F. E. Batten and Dr. James Collier. In the latter paper, besides the observation of seven cases, there is a very comprehensive survey of the whole subject, and an interesting and suggestive attempt to divide the clinical history into three stages; the first being one of slight spastic paraplegia, with slight ataxy and marked subjective sensations in the lower limbs; the second, a stage of severe spastic paraplegia, with marked anæsthesia of the trunk and limbs; and the third, a stage of complete flaccid paraplegia, absent knee jerks, absolute anæsthesia, rapid wasting, paralysis of sphincters, and œdema of lower limbs. It may be said at once that there is no doubt that such a clinical history is more or less closely descriptive of a large number of cases, as it is, *e.g.*, of that of which I have given the particulars at the commencement of this paper. It is not, however, applicable to all.

In my earlier paper, in 1898, I referred to an extremely important paper by Dr. James Putnam, of Boston—a paper which has not, I venture to think, received in this connection anything like the attention it merited. It was published in 1891, and described "a group of cases of system sclerosis of the spinal cord associated with diffuse collateral degeneration, occurring in enfeebled persons past middle life and especially in women." The symptoms generally consisted in progressive impairment of both sensory and motor functions of all four extremities, associated after a time with general wasting of muscles and emaciation. The fatal cases ran their course in about two years; in one case four years. Incoördination of movement was usually present. In several the knee-jerk was exaggerated and ankle clonus was present. In some obstinate diarrhœa was a prominent symptom. The anatomical changes found in the cord were extensive sclerosis of lateral and posterior columns and disintegration of the cells in the gray matter. Dr. Putnam also gave a short clinical account of four other cases with similar symptoms. In all there was great weakness with emaciation, and in some, at least, profound anæmia. In two the knee-jerk was absent.

In my earlier paper I expressed the opinion that notwithstanding some, as I consider, unimportant differences, these cases of Dr. Putnam's were to be regarded as belonging to the same group as Dr. Bowman's and my own, and also the cases described by Lichtheim and Minnich. I pointed out the clinical conditions in all as characterised by the gradual onset of symptoms, weakness and paraplegia, the association of these with, in all cases presumably, in some certainly, a greater or less degree of anæmia, in some profound anæmia, the variability of the reflexes, in some instances exaggerated, in others diminished or absent. And the actual changes found in the cords presented at least very striking resemblances—the same widespread sclerosis affecting the same parts of the cord and the same tendency to limitation of this change to similarly situated comparatively small areas in the lumbar region. Dr. Putnam, however, had found changes in the gray matter which neither Dr. Bowman nor myself had detected in our cases. On the other hand, in many of the cases since described such changes have been distinct.

A somewhat futile discussion has taken place in reference to the question whether these changes are associated with *pernicious* anæmia.

There seems to be no definite agreement as to the clinical condition and state of the blood constituting this disease. But there seems to be little doubt that in some of these cases with cord changes, the condition has been one in which the blood changes supposed to be characteristic of pernicious anæmia have been present, and in which the organs after death have given the reactions supposed to be characteristic of this disease. But it is equally certain that all the cases with these cord changes have not been cases of pernicious anæmia. In the paper already alluded to I pointed out the association with profound anæmia, so profound as to be associated in Dr. Bowman's case and in one of mine with retinal hæmorrhages, and it must be acknowledged that in most of the cases anæmia has been a prominent feature. In one of Risien Russell's cases the condition of bad health had been apparently induced thirteen years before admission by a severe hæmorrhage occurring at a confinement. It is interesting to note that in Dr. Bowman's case the first symptoms had been induced also by a severe loss of blood. Yet although in the vast majority of recorded cases anæmia has been distinct, and in many cases profound, there have been cases in which anæmia was not so marked, but in those cases malnutrition was a distinct feature. Further, the cases are to be divided into two, it may be, somewhat indefinite groups, according to the duration of the clinical history. In some, and usually, I believe, those are the cases in which anæmia is marked, the course of the disease is rapid, and the fatal termination ensues in a few months from the onset. In others the progress of the disease is slower, and a year, or even several years may elapse before death ensues. Such a distinction was foreshadowed in my first paper, Dr. Bowman's and my own cases falling into the first group, and Dr. Putnam's into the second. The distinction has been drawn more definitely in a monograph by Bastianelli, and it may be as he is inclined to believe, that anatomical as well as clinical differences distinguish the two groups. But it is scarcely correct to draw an anatomical distinction between the two in reference to the comparative affection of the lateral columns. Bastianelli says that in the first or rapidly fatal group the lateral columns are comparatively slightly affected; in the second or more chronic group the affection of the lateral columns is severe. The case I have just described disproves such a view, for it is not easy to imagine a more complete affection of the lateral columns than exists in this case in the dorsal region, and the clinical history of the case extended over only nine months. And Putnam points out that in one of his cases in which the symptoms extended over as long as thirteen years the spinal lesions were comparatively slight.

The question has also been raised whether these changes in the spinal cord are the result of vascular lesions. I have already expressed the view that, to a slight extent only, they probably are. In certain cases a hyaline degeneration of the vessel wall has been described, yet this has not been constant, and the changes have not been most marked in the vessels related to the most markedly sclerosed areas. Yet I think it possible that small haemorrhages may occur from vessels in the cord as they do from retinal vessels, and give rise to small sclerotic foci, which coalesce with the larger areas which arise from some other causes. In one of the cases in Drs. Russell, Batten and Collier's paper there was a hæmorrhage into a posterior root ganglion, and in one of my cases there was a distinct unilateral difference between the sclerosis in the upper cervical and lower cervical segments. Yet it is only right to say that Russell does not share this view as to hæmorrhages accounting for any of the sclerosis.

In trying to account for this widespread sclerosis in these cases we seem to me to be driven back on the toxic theory, first promulgated by Lichtheim, and under such a view the sclerosis and the anæmia might both be the results of the same toxin. What the supposed toxin is and where it is generated we do not know, but we do know of analogous conditions in which a chemical toxin does give rise to analogous cord changes. In diabetes, *e.g.*, we have a toxic blood state, and the observations of Williamson have firmly established the presence of sclerosis in the posterior column of the cord in many cases of diabetes. In pellagra also, a disease arising from eating unripe maize or even from drinking spirit made from such maize, we have changes in the cord strikingly similar both in extent and distribution to those present in the cases under notice. The symmetrical character of the sclerosis also is in favour of such a view, and the recent important work of Adami, Hunter and others is in favour of it.

One point remains to be considered, viz., the relation of the cases of ataxic paraplegia of Sir William Gowers to the cases mentioned above. Ataxic paraplegia is, of course, a clinical name, and it must be confessed that many of the cases above described are at some stage of their clinical history in a state of paraplegic weakness associated with ataxy. In Gowers' original cases the history was one spread over a considerable number of years as a rule; so is the history in many of Putnam's and Dana's cases, which one might call the American group. It is interesting that, in reference to a case published in BRAIN (vol. xiii., p. 356) by Dr. Michell Clarke of Bristol, as one of ataxic paraplegia, the author of that paper should himself have acknowledged to me after the publication of my first paper that he had gone into the matter again and re-examined his sections, and he had no doubt the case was one of the class I alluded to. Personally, I should be inclined to think that in this large class of cases, best known under the curiously hybrid name of "subacute combined sclerosis," there are very many varieties of the same disease, yet that the disease is essentially a unity, just as much as is tabes dorsalis, and that most, if not all, cases of ataxic paraplegia are cases of this disease. It is also a point of great interest that nearly all the cases described by American observers—Putnam, Dana and Taylor—should have run a comparatively long clinical course. It is possible, however, that with the increased attention now devoted to this disease it may be found that they have the acute variety also in America, and that we have the chronic variety in this country more frequently than has been supposed.

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THE EFFECTS OF TOTAL TRANSVERSE LESION OF THE SPINAL CORD IN MAN.

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THE argument of this paper is that as the result of total transverse lesion of the spinal cord in man, not only are the knee-jerks and other deep reflexes permanently abolished in the region supplied by the caudal segment of the divided spinal cord, but in addition, the muscles waste and lose their faradic excitability, and the sphincters lose their tone, the only sign of self-action remaining in the isolated part of the spinal cord being the occasional presence of certain of the skin reflexes in much reduced degree. These phenomena occur in the absence of any recognisable structural change in the ventral horn cells, ventral roots and peripheral nerves of the paralysed region. That muscular wasting, loss of faradic excitability and loss of sphincter tone may occur in cases of transverse lesion of the cord was long ago pointed out by Sir William Gowers (10), but in his opinion the presence of these signs definitely proved the co-existence of concomitant or consecutive changes in the lumbo-sacral enlargement.

Attention is drawn to the fact that when the knee-jerks have disappeared in cases of progressive transverse lesions becoming total transverse lesions, for a certain time after such disappearance the knee-jerks may be again obtained subsequent to long-continued faradisation of the lower limbs. In other words, the lumbo-sacral centres in man have not sufficient self-action to maintain the muscular tone when severed from the higher centres, but they may be temporarily roused by peripheral stimulation into such a higher state of activity as will allow the knee-jerk to be obtained. The importance of this fact, here recorded for the first time, in proving that the loss of knee-jerk is due to isolation from the higher centres and not to any concomitant injury to, or consecutive structural changes in, the lumbo-sacral centres is obvious.

These observations are based upon a considerable number of cases of complete and partial transverse lesions of the spinal cord which have been treated in the wards of the National Hospital, and my best thanks are here expressed to my colleagues upon the Staff of that hospital for permission to make use of the notes. I am indebted to Mr. Stansfield Collier for permission to observe and record two cases from St. Mary's Hospital.

The larger number of the cases were examined pathologically, while in several cases operative interference permitted the nature and extent of the lesion to be determined.

The cases were those of wounds of the spinal cord, fractured dislocations, compression paraplegia and acute transverse myelitis.

Brief reference is necessary in this place to the results of experimental transection of the spinal cord in animals and to the very wide difference of these from the effects of transverse lesions in the human subject.

In the cat and in the dog spastic paralysis results from transection, the deep reflexes are exaggerated and the most complicated reflex actions can be elicited, the "scratch" reflex, for example. Sometimes the transection is followed by temporary diminution or abolition of the deep reflexes, which lasts for a few minutes only.

In the higher apes transection of the cord is followed by spastic paralysis, but the spasticity is not nearly so marked as in the lower mammals. Complicated reflexes are not obtained. The knee-jerks are often absent for several days after the transection, but sometimes they return directly the effects of the anæsthetic has passed off. In these animals the autochthonicity of the isolated part of the spinal cord seems to be greater a few weeks after the transection than at later periods, and this capacity for self-action lessens as time elapses. The term "isolation-alteration" has been applied to this slow process of physiological deterioration. The tendency towards temporary loss of the knee-jerk, its occasional permanent abolition, the spasticity little marked and the signs of self-action decreasing with lapse of time, make the effects of transection in the higher apes easily comparable with the effects of transverse lesion in man, the result being intermediate, as it were, between the spastic paralysis with exaggerated knee-jerks occurring in the dog and the flaccid paralysis with absent knee-jerks occurring in man.

I wish to draw particular attention to Professor Sherrington's description of isolation-alteration in monkeys (18) in connection with the loss of sphincter tone, muscular wasting, loss of faradic excitability and trophic changes, which gradually follow upon the occurrence of a total transverse lesion in the human subject. While such isolation-alteration is gradual with slow steps in the higher apes, it is gradual with rapid strides in man, and is manifest a few days after the lesion has become total.

In man it would appear that the disappearance of the knee-jerk is not synchronous with the occurrence of the lesion, since the knee-jerk has been repeatedly obtained in decapitated criminals (19), and it has been noticed to persist as long as ninety seconds after execution (20).

Since it was first pointed out by Dr. Bastian (4) that a total transverse lesion of the spinal cord entailed abolition of the deep reflexes, the effects of such lesions have been a matter of much controversy, the opinions of many authorities having been doubtless influenced by the effect of experimental transection in animals. At the present time it is almost universally accepted that a total transverse lesion results in flaccid paralysis, with loss of the deep reflexes, but it is also held that the knee-jerks may return after a lapse of time, and, further, several published cases of total transverse lesion in which the knee-jerks have persisted are accepted as undoubted facts.

As this paper deals only with the results of personal observations, I have considered it expedient to make but few references to the extensive literature of the subject. It is allowable, however, to remark that writers upon this subject have not worked from the same standpoint as to what is a total transverse lesion, and, unfortunately, in some of the cases which have been published as examples of total lesion with persistent knee-jerks the totality of the lesion admits of doubt.

Pathologically, a transverse lesion of the spinal cord may be deemed total:—

(1) When the spinal cord is actually severed.

(2) When serial longitudinal sections through the region of the lesion stained by Weigert's method show no myelinated fibres passing through the length of the lesion, and when special methods for staining axis cylinders show a similar absence of these structures in the length of the lesion.

(3) When serial transverse sections through the region of the lesion, stained by a nuclear stain, show the whole area of transverse section to be necrotic.

The size of the spinal cord in the region of the lesion is no criterion of the extent of damage. Extraordinary reduction of the size may occur in pressure paraplegia with complete preservation of sensibility, as in Case 14.

Clinically, a physiological total transverse lesion may be considered to exist when there is absolute loss of sensibility, complete flaccid paralysis and loss of the deep reflexes.

Such a complete physiological abrogation of the functions of a transverse area of the spinal cord may exist without actual death of the elements involved, and these elements may recover more or less completely, as is frequently the case in pressure paraplegia and lesions involving evascularisation of a transverse area of the spinal cord.

When there is complete degeneration of all the elements of a transverse area of the spinal cord there can be little doubt that sufficient continuity for the maintenance of the spastic state and for the return of the knee-jerks may be re-established by the regeneration of conducting paths. That such regeneration does occur is strongly suggested by the conditions found in Cases 2 and 3, and by the behaviour of some cases of acute myelitis, which begin to show marked improvement long after the onset. Immediate suture experiments upon the spinal cords of animals have shown that regeneration is possible in the spinal cord, and the researches of Bielchowski (5) argue strongly that it is no rare event as a sequel to lesions of the human nervous system.

In a large number of cases here considered the onset of symptoms has been gradual; a progressive lesion having at first produced partial, and later total, abrogation of the function of the involved part of the spinal cord. It is therefore of interest to consider the general order in which symptoms appear.

In another larger group of cases the onset has been sudden or rapid (fractured dislocation and acute myelitis), and the clinical picture of total physiological transection has been produced at once or early, but various degrees of recovery have subsequently occurred. The general order of regression of symptoms is therefore considered.

In a few cases the clinical picture of total transverse lesion has changed to one of incomplete lesion at a period so remote from the occurrence of the lesion and under such circumstances as to suggest strongly that the conductivity through the region of the lesion had been restored by the regeneration of the nerve-fibres.

With gradual lesions the order of appearance is practically constant as follows: motor paralysis and spasticity, equilibratory anæsthesia, sphincter paralysis, thermanæsthesia and analgesia, tactile anæsthesia, flaccidity with loss of the deep reflexes, progressive muscular wasting, progressive lowering of faradic excitability, progressive loss of sphincter tone. The motor paralysis and spasticity develop slowly and with even steps. Sphincter paralysis is variable, sometimes appearing long before any skin sensory loss is apparent, sometimes being delayed till skin sensory loss is marked. Loss of skin sensibility from increasing lesion, when once apparent, progresses much more rapidly than does the motor paralysis, and it would appear that sensibility to pain and temperature is always impaired earlier and to a greater extent than is sensibility to touch.

The place of appearance and the spread of sensory loss upon the limbs and body seem to follow one of two definite orders. Taking, for example, lesions of the lower half of the thoracic region: in the larger number of cases the sensory loss first involves the skin in the order of the length of the sensory neurones supplying it. The soles of the feet first become asentient, then the legs, and, later, the thighs; but when the anæsthesia reaches the trunk it extends upwards in segmental order.

In a smaller number of cases the sensory loss is first manifest over the most caudal segmental areas and extends upwards in segmental progression. It involves the segmental areas indirectly as their distance caudal of the lesion, the lowest sacral first, then the upper sacral, and, later, the lumbar areas.

The change from the spastic state to the flaccid state takes place rapidly in from twenty-four hours to a week, according to the acuity of the lesion, and is accompanied or shortly followed by loss of the deep reflexes. It never occurs till the sensory loss is complete over the lower extremities. Muscular wasting, lowering of faradic excitability and loss of sphincter tone commence with the onset of the flaccid state, and are progressive so long as the physiological transverse lesion remains total.

When there is recovery from the total physiological transverse lesion, the first sign of such recovery is the return of the knee-jerk and a gradual change from the flaccid to the spastic state. Sensibility returns in the order, touch, pain, temperature; and the lowest sacral segments usually become sentient before the others. The first signs of such return is often that the patient becomes conscious of the act of micturition, or of catheterisation, or of distension of the bladder during the routine lavage. In one case sensibility returned first in the feet.

Voluntary power returns on the flexors earlier than on the extensors, flexion of the toes being almost invariably the earliest voluntary movement possible.

In cases where recovery is complete the plantar reflex does not resume the flexor type till some time after the patient has completely recovered the use of the lower limbs, the extensor response being invariably the last of all spastic signs to disappear.

In the few cases where it is suggested that regeneration had taken place, the return of the knee-jerk, followed by a slow change from the flaccid to the spastic state, was the only constant feature. Further recovery was slight and its nature inconstant. In one case slight voluntary movement of toes of the left foot first returned, but there was no return of sensibility. In another case the patient became conscious of having his joints moved.

SENSIBILITY.

The nature of distribution of sensory loss has been already discussed in the consideration of the onset of symptoms.

A noticeable feature of these cases has been the extreme rarity of any detectable zone of hyperæsthesia in the region immediately above the lesion. The writer has only met with it twice—once in connection with a cervical lesion where the third and fourth cervical root areas were distressingly sensitive (this case was not examined pathologically), and once in connection with a low thoracic total lesion where meningeal new growth was obviously involving several nerve roots above the level of the lesion of the spinal cord (Case 15).

Where pressure lesions exist the onset of complete anæsthesia does not mark an irrecoverable stage of the lesion, but the pressure must be relieved soon if recovery is to take place. Complete recovery occurred in Case 4, where a myxoma pressing upon the ninth dorsal segment was removed four days after the anæsthesia had become complete. Again, anæsthesia and complete motor paralysis are no proofs that a lesion is total unless flaccidity and loss of the deep reflexes co-exist.

MUSCLES.

Most important evidence as to whether a lesion is total or not is to be found in the condition of the muscles. When the lesion is total the muscles become at once flaccid ; they waste rapidly and their faradic excitability lessens, rapidly at first, then more slowly, and finally it may be lost. Where the lesion is not total slight wasting of the muscles occurs, but the tone is increased and the slight lowering of faradic excitability that may be met with seems to vary as the lesion occurs, rapidly or gradually. In a case of destruction of several of the upper thoracic segments by a wound, the muscles of the legs showed the faintest response to strong faradic stimuli on the fifth day after the injury. In the case of myxoma pressing on the ninth dorsal segment, the strongest obtainable faradic stimulus only caused a faint reaction on the third day after the onset of flaccidity.

The muscular wasting affects all the voluntary muscles supplied below the lesion equally, and, in the course of time, it may come to be as severe as that which occurs in cases of anterior poliomyelitis, with complete involvement of the lower extremities, and, in cases of destruction, of the cauda equina.

The lowering and ultimate loss of faradic excitability to stimuli applied through the skin is not accompanied by initial increase of galvanic excitability, nor by any marked increased reaction to "make" stimuli at the anode. It is important to bear in mind in this connection that an atrophied muscle always responds to faradic stimuli applied directly to the muscle, whatever be the cause of the muscular atrophy, provided that muscular tissue still exists.

If, for example, a pair of needle electrodes be introduced into muscles which have been for years completely paralysed as the result of anterior poliomyelitis or nerve section, a single faradic shock of moderate intensity produces a local contraction of the muscle in the region of either electrode, and thus contraction is readily detectable, owing to the lever movement of the electrode upon the point where it pierces the skin. This local contraction may be that of the intrafusal fibres of the muscle spindle, which do not atrophy as the result of section of the motor neurone supplying the muscle.

It has been argued that the muscular wasting and loss of deep reflexes which occur in total transverse lesions of the spinal cord are the result either of coincident damage to the lower motor neurones of the lumbo sacral region of the cord, or of consecutive structural changes in these elements.

These explanations are directly negatived by the fact that the most thorough microscopic examination of the lower motor neurones in many of the cases failed to show the least departure from the normal in any part of their course, the richness of the tigroid substance and the distinctness of its arrangement, and of the cell processes, affording excellent demonstration of normal ventral horn cells in a state of rest.

Exception must be made in the case of the cells of Clarke's nucleus and the equivalent sacral nucleus, which invariably show marked signs of degeneration after transection of the cord. This fact was first pointed out by Schäfer (14), and it has been repeatedly confirmed by other writers.

Brasch has recently found degeneration both of the ventral horn cells and of the lower motor neurones in cases of total transverse lesions, with loss of the deep reflexes. He considers that this is the result of tertiary degeneration (isolation atrophy), and, in his opinion, the presence of such degeneration explains the flaccid paraplegia and loss of deep reflexes. The immediate onset of flaccid palsy and the immediate abolition of the knee-jerks, however, cannot possibly be the result of any such degeneration, nor can the rapid return of spasticity and of the deep reflexes, which occurs when pressure is relieved, after a physiological total lesion, be explained upon such grounds. If tertiary degeneration sufficient to cause flaccidity and abolition of the knee-jerks ever occurred, its clinical signs would be that after a total lesion the muscle tone and deep reflexes would gradually disappear, and this does not occur. Francotte, Leyden and Marinesco have also found changes in the ventral horn cells, Bischoff and Bartels have found the posterior roots of the lumbar region degenerated, while Oppenheim and Siemerling have found changes in the peripheral nerves of the lower extremities.

Slight abnormality of the ventral horn cells was met with in several cases where malignant disease, suppuration, or pyrexia had existed, but such changes were not greater than those which may occur in similar conditions apart from any spinal cord lesion.

Clinical evidence strongly supports the argument that muscular wasting, lowering of faradic excitability, and loss of deep reflexes under consideration, are the result of isolation of the lumbo-sacral region of the cord from the higher centres above. For in cases of complete recovery from a physiological transverse lesion, the writer has seen these phenomena develop rapidly and disappear with such rapidity after the removal of the cause of the lesion as to render any other explanation difficult. Further, in cases of acute transverse myelitis and fractured dislocation, where there is at first a total physiological lesion, these symptoms appear to disappear as soon as some conductivity through the lesion is regained.

Again, in those cases where it is submitted that regeneration had taken place, these signs disappeared. Most conclusive evidence in support of this argument is that for a few days after the occurrence of a total transverse lesion strong stimulation of the lower extremities often causes a temporary return of spasticity and of the knee-jerk.

The microscopic appearance of the wasted muscles is peculiar. The muscle fibres are greatly and regularly reduced in size (intrafusal fibres excepted). The transverse structure is ill-marked, but usually apparent; the nuclei are not increased. Fatty changes and vacuolation are not present, and there is only slight connective tissue proliferation and moderate periarterial thickening. These changes are strikingly different from those seen in muscles where the supplying neurone has been injured.

It may be remarked that Dr. Sherrington (17) has shown that certain muscles, notably those supplied by the third and sixth cranial nerves, show few histological changes as the result of section of their motor nerves, and that these muscles possess no spindles.

THE DEEP REFLEXES.

A total transverse lesion of the spinal cord, whether physiological or anatomical, causes abolition of the deep reflexes below the lesion. The return of the deep reflexes at any subsequent period is significant that the lesion has not remained total, and that conductivity through the region of the lesion has again become possible either from recovery of elements of which the function has been in abeyance for some time and has been resumed, or from the regeneration of nerve fibres.

It would appear from the previously mentioned observations upon decapitated subjects that the deep reflexes may be retained for some short time after the occurrence of a total lesion.

The writer has had the opportunity of examining three cases of sudden transverse lesion within two hours of the occurrence, and has always found a condition of complete flaccidity with absent deep reflexes.

Where progressive lesion passes on to total lesion the writer has been able to demonstrate repeatedly that for some few days after the occurrence of the flaccid state, with loss of deep reflexes, strong and continued stimulation of the skin or nerves of the lower extremities has the effect of rousing the sleeping lumbo-sacral centres to such a degree of activity that their self-action is expressed by a temporary return of the deep reflexes and in some cases the presence of foot clonus.

The procedure was carried out as follows: A sterilised gilt needle electrode was passed deeply into the region of the external popliteal nerve below the head of the fibula. A similar needle was passed deeply into the calf. The two electrodes were connected with a strong interrupted current, which was allowed to act from five to twenty minutes, after which the conditions of the muscular tone and of the deep reflexes were tested.

It was found that if the state of flaccidity with absent deep reflexes had not existed more than three days the knee-jerks were easy to bring back. If this condition had lasted longer than three days, as each day elapsed longer stimulation was required; the knee-jerks when obtained were more feeble, and they disappeared sooner. While after ten days prolonged stimulation was in no case followed by return.

The deep reflexes were obtainable for from five to fifteen minutes after the cessation of stimulation. Most marked immediately after the current ceased, they gradually became less, their elicitation lost its certainty and they disappeared.

Of special interest in this connection was the observation that in two cases of long-standing total transverse lesion, which are submitted as cases in which regeneration had taken place, and in which the knee-jerks returned late, the latter were first obtained after the patient had been subjected to faradism of the lower extremities in the way above described.

THE SKIN REFLEXES.

These appear to be invariably abolished in severe transverse lesions with the exception of the plantar reflexes. The latter may be increased where the lesion is incomplete, where some recovery from a total physiological transverse lesion has occurred and when regeneration has taken place.

When the lesion is total, reflex action from the skin is much depressed, and it has been stated by several authorities that it disappears. It may be unobtainable directly after the occurrence of a sudden lesion, but the writer has found it present in three cases of sudden total lesion within two hours of the injury. The only reflex obtainable in the cases was a feeble response of the extensor type to stimulation of the sole of the foot.

Where life is prolonged with a total transverse lesion, it seems probable that all signs of reflex action slowly disappear.

VISCERAL REFLEXES.

Priapism has been met with rarely in this series of cases, only in connection with partial lesions, and never where a total lesion existed. In the writer's opinion its presence signifies that the lesion is partial.

The condition of the sphincters in this series of cases did not accord with the classic description.

After the occurrence of a total lesion there may be temporary retention, and there may be reflex evacuation for a time, but the persistence of these phenomena seems to indicate that the lesion is not total. Constant dribbling with a small bladder has been the rule.

The anal and urethral reflexes have been repeatedly examined in these cases, and reflex evacuation tested by the application of cold to the skin supplied by the lower sacral segments, and it would appear that these reflexes and the sphincter tone are present directly after the occurrence of a total lesion, and that they gradually lessen till the orifices become patulous; reflex evacuation soon becomes difficult or impossible to bring about, active retention by the sphincters ceases and constant escape occurs, and the bladder slowly contracts and its lavage becomes a matter of difficulty.

The self-action of the centres of the bladder and rectum seems to be retained to a much greater extent in young children, and in this connection it is of great interest that in a case of a child of 6 weeks old, which has been published by Dr. Beevor, the writer had the opportunity of demonstrating spontaneous evacuation of the bladder, resembling both in periodicity and otherwise reflex evacuation, notwithstanding the fact that the whole spinal cord below the sixth cervical segment was destroyed and possessed no recognisable living nerve elements. A similar phenomenon has been recorded as occurring in very young animals after complete destruction of the lumbo-sacral enlargement.

TYMPANITIS.

Abdominal distension has been met with in a few cases where the lesion has involved the lower cervical and upper thoracic regions of the spinal cord.

It makes its appearance within a few days of the occurrence of the lesion, and if the patient lives it soon disappears. It seems never to occur as a late symptom of transverse lesion of the cord, and in this series it has been present only in cases of fractured dislocation.

Œdema of the lower extremities was noticeable as an early and evanescent symptom in a few cases of severe or total lesion. It is prone to occur when malignant cachexia co-exists or when toxæmia and pyrexia are present.

Trophic changes have been very little in evidence in this series of cases, but slight changes in the skin and nails have been the rule where severe lesions have been long survived.

Bedsores do not occur if the skin is properly attended to. When present at the time of admission of the patients they have healed in many instances, where they had not reached an advanced stage.

Changes in the joints were not met with apart from rest adhesions.

POSSIBLE CLINICAL UTILITY OF THE RETURN OF THE KNEE-JERKS AFTER FARADISATION AS A PHYSICAL SIGN.

The writer submits that in some cases of flaccid paraplegia the return of the knee-jerks subsequent to faradisation of the lower extremities may render it possible to determine whether there is implication of the lumbo-sacral enlargement and its nerve roots or not. For example, a case of progressive paraplegia from a lesion with its upper limit in the low thoracic region while under observation passes from the spastic to the flaccid state. If, soon after the onset of flaccidity and the loss of the knee-jerks, the latter return temporarily as the result of faradisation of the legs, it is certain that the lumbo-sacral enlargement and its roots are not involved in the lesion. But, if no such return can be brought about, it argues strongly that the lumbo-sacral enlargement or its nerve roots are so involved.

Case 15 is cited as a case in point, for many attempts were made to elicit the knee-jerks after faradisation, but without success, and the autopsy proved that the lumbar roots were seriously involved in the meningeal thickening.

It is of considerable interest that in the cases here recorded complete recovery has followed the removal of pressure where the signs of total abrogation of function of a transverse region of the cord, namely, complete flaccid paraplegia, total loss of sensibility and loss of deep reflexes, have existed for as long as seven days. The recovery has been so rapid and so complete as to make it certain that pressure may produce complete physiological abrogation for many days without necessarily entailing degeneration of the nerve elements. The onset of the flaccid state in compression paraplegia is a sign which indicates that operative interference for the relief of pressure must be undertaken at once if it is to be of any avail, for the total physiological abrogation from compression signifies the presence of serious evascularisation, and the inevitable sequel of such ischemia —irreparable degeneration—cannot be long delayed.

APPENDIX OF CASES.

Where several cases of the same clinical type have been examined, details of one only is given. Two cases similar to Case 3 have been examined, and many cases of similar nature to Cases 5, 6, 10, 11, 12 and 14 have been used in this communication.

Case 1.—*Destruction of several segments of the upper thoracic cord by injury.*

A man, aged 52 years, was admitted into St. Mary's Hospital under the care of Mr. Stansfield Collier in November, 1898. He had fallen from a considerable height, striking his back upon a rough projection. When examined one hour after the injury he had complete flaccid paralysis and complete anæsthesia below the fifth dorsal level. The knee-jerks were absent; a sluggish extensor plantar reflex was obtained on both sides.

The spinal cord was immediately explored. The posterior arches of several of the upper thoracic vertebræ were fractured, the fragments having been driven into the spinal canal. The spinal cord was found to be completely severed above the fifth dorsal segment, some two inches of its length being destroyed. Death occurred four days later. On the day of the injury there was retention of urine. On the following day the sphincter tone was present, but poor, and there was reflex evacuation. Subsequently the sphincters were almost toneless, and dribbling occurred. There was slight tympanites. The condition of paraplegia remained the same till death, and the extensor plantar reflexes persisted. The muscles of the lower extremities wasted markedly, and on the day before death only a slight contraction could be obtained with a strong faradic current.

Remarks.—The totality of the lesion was demonstrated and the symptoms were classic. Of special interest were the rapid failure of the sphincter tone and the early lowering of faradic excitability.

Case 2.—*Fractured dislocation, total lesion, probable restoration of conduction by regeneration. Autopsy.*

G. M., aged 46 years, was admitted into the National Hospital under the care of Dr. Tooth on August 25, 1899. As the result of a fall twelve days before admission a fractured dislocation of the ninth dorsal vertebra had occurred.

There was complete flaccid paralysis and complete anæsthesia below the umbilicus. There was retention of urine, with occasional reflex evacuation of the bladder, for some weeks after admission; subsequently the sphincter tone became much reduced. The muscles of the legs were much wasted, and there was no response to faradism applied through the skin. Faradism applied by means of needle electrodes introduced into the muscles produced a slow local contraction.

The knee-jerks were absent; there was no foot clonus. There was a brisk plantar reflex of the extensor type.

Though this patient was repeatedly examined, the knee-jerks were never obtained until January 12, 1900, when five months after the accident the right knee-jerk was found present after the application of continuous strong faradism to the right leg. It persisted for about five minutes after the stimulation was discontinued, and then disappeared. The left knee-jerk was not obtained.

On the following day faradism was applied to both legs for about twenty minutes, after which both knee-jerks could be obtained, and they persisted for about fifteen minutes, right and left.

Two days subsequently, the knee-jerks being unobtainable, the experiment was repeated, and the knee-jerks appeared and persisted. Feeble at first, they gradually became brisk, and rigidity made its appearance in the legs. The reflex action of the lower extremities became exaggerated, but no sensibility returned. A year later he was still completely anæsthetic below the umbilicus, but he could move the toes of both feet to a very slight extent. The knee-jerks remained brisk. He died of cerebral thrombosis on December 15, 1900.

The necropsy showed that the spinal cord was reduced to the size of a quill for a length of about 6 cm. in the lower part of the tenth dorsal segment. There was practically no bony deformity of the vertebral canal, the dislocation which caused the damage to the cord having presumably become immediately reduced.

Longitudinal sections through the region of injury stained by the Weigert-Pal method showed that there were a considerable number of fine fibres passing through the lesion, and that there was nerve continuity between the upper and lower limits of the lesion. The appearance and irregularity of these fibres suggested that they were fibres which had regenerated.

The persistence of signs of a total lesion of the spinal cord in the absence of any pressure upon the cord for five months, and the subsequent return of spasticity and slight voluntary power, suggest strongly that regeneration had taken place. This suggestion was rendered probable by the microscopic examination of the region of the lesion.

The return of the knee-jerks after prolonged peripheral stimulation is noteworthy.

Case 3.—*Fractured dislocation, total lesion, return of knee-jerks after faradisation six months later. ? Regeneration.*

J. R., aged 38 years, was admitted into St. Mary's Hospital under the care of Mr. Stansfield Collier in September, 1899. He had fallen a distance of ten feet, striking his head and left shoulder.

When admitted there was complete flaccid palsy and complete loss of sensibility to all forms of stimulation below the arms. The knee-jerks were absent. The plantar reflexes were extensor. Priapism and retention of urine occurred during the twenty-four hours following the accident. There was slight bilateral ptosis, and no light reaction could be obtained.

He remained in the same condition, except that the sphincter tone diminished markedly, and the muscles of the lower extremities wasted considerably and their faradic excitability became much reduced.

On March 25, 1900, after prolonged faradisation of the lower extremities, the right knee-jerks were obtained.

Faradisation was frequently applied, but the left knee-jerk was not obtained till three weeks later. Both knee-jerks persisted, and some degree of spasticity slowly returned. It is submitted that in this case the return of the knee-jerks was coincident with the restoration of some conduction through the lesion by regeneration.

Case 4.—Pressure paraplegia. Total physiological lesion, knee-jerks obtained after faradisation for three days subsequent to the onset of the flaccid state. Operation. Recovery.

G. P., aged 36 years, was admitted into the National Hospital under the care of Dr. Buzzard on October 13, 1898. Symptoms of pressure upon the spinal cord first appeared in July, 1898, and had become progressively more marked.

When admitted there was absolute analgesia and thermanæsthesia, with marked tactual impairment over the whole of the right lower extremity, and over the tenth, eleventh and twelfth dorsal and first lumbar areas of both sides. Over the left lower extremities there was marked loss to pain and temperature. There was spastic paralysis, and all movements of the lower extremities were possible, but were very feeble. There was slight dysuresis.

After admission the paralysis rapidly became absolute, and retention of urine occurred. Subsequently the anæsthesia became absolute below the ninth dorsal level. The knee-jerks gradually diminished, and foot clonus was no longer obtainable. On November 14 the paralysis became flaccid, the knee-jerks were not obtained on reinforcement, and complete incontinence of both sphincters set in, and the muscles of the lower extremities began to waste rapidly.

For the next four days the knee-jerks were obtained after strong faradisation of the legs.

As each day elapsed stronger stimulation was necessary for the elicitation; the knee-jerk, when elicited, was more feeble and lasted a shorter time after stimulation. On the fifth day the knee-jerks could not be elicited, even after thirty minutes' strong faradisation. Two days later the muscles reacted only to the strongest faradic stimuli applied to the skin.

On November 22 Sir Victor Horsley performed laminectomy, and removed an intra-thecal fibro-myxoma about the size of an acorn which was attached to the tenth left posterior root, and which was pressing into the posterior aspect of the cord.

The patient made an uninterrupted recovery. Five days after the operation he could feel catheterisation, and the knee-jerks returned. Subsequently sensibility returned in segmental order from below up. The legs became markedly spastic. Voluntary power was first noticed on December 26, and subsequently it returned rapidly. On January 9, 1899, there was considerable loss of sense of position at all joints in the lower extremities notwithstanding that common sensibility had completely returned.

He was able to resume work as a farm steward in the summer of 1899.

Case 5.—Total lesion. Return of the knee-jerks, produced by peripheral stimulation, for a few days after the lesion had become complete.

A. S., aged 50 years, was admitted into the National Hospital on October 25, 1898, under the care of Dr. Buzzard, suffering with cancer of the dorsal vertebræ, secondary to a scirrhus of the left breast which had been removed. There was complete flaccid paraplegia and complete loss of sensibility below the tenth dorsal level. The knee-jerks were absent and the plantar reflexes were extensor.

There was complete incontinence, and the sphincter tone was only just present.

For six days after admission strong faradisation of the legs by means of needle electrodes was followed by a return of the knee-jerk, which persisted in diminishing intensity at the longest for five minutes.

Subsequently the anæsthesia extended as high as the seventh dorsal level. The faradic excitability of the muscles of the lower extremities became progressively lowered, and finally lost to stimuli applied to the skin. She died on January 10, 1899. The pathological examination was made by Dr. Risien Russell. The spinal cord was reduced to the size of a crow-quill in the seventh dorsal segment, and its substance was semi-diffluent at this point. There was complete evidence of total transection. The caudal enlargement and its nerve roots were normal.

Case 6.—Pressure paraplegia, total physiological lesion. Operation. Recovery.

C. G., aged 13 years, was admitted into the National Hospital under the care of Dr. Bastian on May 20, 1900.

His symptoms began six weeks before admission with severe pain in the left side, followed by weakness of the left leg.

On admission there was complete spastic paraplegia. There was marked loss of pain and temperature below the sixth dorsal level. Very slight and indefinite tactile impairment over the legs. There was slight dysuresis. The knee-jerks were exaggerated, double foot clonus was present and the plantar reflexes were extensor.

On June 15 there was complete analgesia and thermanæsthesia in the paraplegic region, but tactile sensibility was almost perfect. On June 22 there was entire loss of control over the sphincters, with retention. Tactual anæsthesia was very marked. On June 30 the anæsthesia was complete to all forms of stimulation. The knee-jerks were absent, but faradisation of the lower extremities for five minutes caused them to reappear for a few minutes. The limbs were absolutely flaccid. Incontinence became complete. On July 6 Sir Victor Horsley removed a large number of hydatids which were compressing the spinal cord in the region of the sixth, seventh and eighth dorsal vertebræ.

On July 17 the knee-jerks were obtainable.

On August 2 heavy touches could be felt everywhere, and there was some return of sphincter control.

On August 16 the first sign of returning voluntary power was noticed, and thermanæsthesia and analgesia were nowhere absolute. He made uninterrupted recovery.

This case demonstrates the order of appearance and disappearance of the signs of compression. It exemplifies also the return of the knee-jerk after faradism when the lesion is physiologically complete.

Case 7.—*Acute myelitis. Total lesion. Autopsy.*

R. B., aged 56 years, was admitted into the National Hospital under the care of Dr. Risien Russell on October 9, 1901, suffering with acute syphilitic myelitis of forty-six days' duration. For ten days after the onset he had had retention requiring catheterisation, and occasional reflex incontinence; subsequently there was complete incontinence. There was complete flaccid paralysis and complete loss to all forms of sensibility below the eighth dorsal level. The right knee-jerks were never obtained; the left was obtained as a mere flicker for two days after his admission, and was not subsequently obtained. The plantar reflexes were extensor. There was extreme general muscular wasting of the lower extremities, and the muscles only responded feebly to strong faradism. There was almost complete loss of sphincter tone, and there was complete incontinence. Some œdema of the flanks and of the lower extremities was present. There were two bedsores and severe cystitis. He died on October 18, 1901. The pathological examination showed that the lesion was limited to the ninth and tenth dorsal segments. The upper two-thirds of the ninth dorsal segment was entirely necrotic throughout the area of transverse section. No normal fibres nor axis cylinders could be traced through this part of the lesion. The caudal enlargement and its nerve roots were normal.

Case 8.—*Acute myelitis. Total lesion. Autopsy.*

E. C., aged 28 years, was admitted into the National Hospital under the care of Dr. James Taylor on August 12, 1902.

In June, 1892, he was seized with typical acute syphilitic myelitis. He had had retention of urine for a few days after the paralysis set in.

When admitted there was complete flaccid palsy and complete loss of sensibility below the eighth dorsal level. The knee-jerks were absent. The plantar reflexes were extensor in type. The sphincter tone was very feeble. There was complete incontinence. The muscles were much wasted, but responded feebly to faradism. There were marked trophic changes in the skin of the lower extremities; several bedsores and severe cystitis were present. He died on September 7, 1902.

The autopsy was performed six hours after death. The lesion involved the ninth and tenth dorsal segments. The upper half of the tenth dorsal segment was completely necrotic, none of the elements staining with alum hæmatoxylin. Sections stained by the Marchi method and by Weigert's method also showed that there was complete disorganisation throughout the area of transverse section.

The caudal enlargement and its nerve roots were uninvolved.

Case 9.—*Acute transverse myelitis. Total lesion. Clinical aspect after two years.*

J. C., aged 12 years, was admitted into the National Hospital under the care of Dr. Bastian on February 20, 1899, with complete paralysis and complete loss of sensibility in the lower part of the body and lower extremities. The family history was unimportant. He had always been delicate, and had suffered with measles, followed by a long lasting discharge from the right ear.

His illness began on January 1, 1897, when having gone to bed feeling quite well, he woke with severe pains in the back and in both arms. On attempting to get out of bed he was unable to move his legs. In the course of the next day the legs and lower part of the trunk became completely paralysed, and he lost all sensibility below the level of the nipples, and he was unable to pass water. He remained in this condition till his admission. On admission he presented the following signs: The optic discs were ill-defined and woolly in appearance at their margins, as if a moderate degree of optic neuritis had been present at some time. The pupils were normal.

There was absolute anæsthesia to all forms of sensibility below the level of the upper border of the second rib. Sensibility was completely retained over the upper extremities.

There was complete flaccid paralysis below the second rib, except in the diaphragm, which acted well. No intercostal movement occurred, nor any movement of the abdominal muscles nor lower extremities.

The paralysed muscles were much wasted, and showed no response to strong faradism nor to strong galvanism applied to the skin. When needle electrodes were introduced into the muscles a feeble slow local response occurred with both forms of stimuli.

There was absolute incontinence of sphincters, with loss of reflex response.

The knee-jerks were absent, and no signs of reflex action could be obtained from the skin.

The legs were faradised with a strong current for an hour, but no reflex response could be afterwards obtained.

It is submitted that in this case a total transverse lesion of the spinal cord in the dorsal region had occurred, and that the absolute lack of any sign of reactive vitality below the lesion was the ultimate result of transection alone and not of concomitant lesion of the lumbo-sacral enlargement. For the clinical picture in this case was only so far more severe than in many of the cases of total lesion here cited as the longer tenure of life subsequent to the occurrence of the lesion might explain on the ground of progressive isolation—alteration. And in those cases in this series which have been examined pathologically, and which have approached closely to the above case in clinical aspect, no changes have been found in the lumbo-sacral enlargement of the cord which would account for its loss of function.

Case 10.—*Acute myelitis, recovering.*

A. E., a laundress, aged 20 years, was admitted into the National Hospital under the care of Sir William Gowers on

September 16, 1898, suffering with acute syphilitic transverse myelitis of six days' duration. There was complete flaccid paraplegia and absolute loss of sensibility below the tenth dorsal level. The knee-jerks were absent; the plantar reflexes were of extensor type. There was complete incontinence, and the anal sphincter was almost toneless. The muscles showed distinct lowering of faradic excitability. The knee-jerks returned on the sixteenth day of the illness, and some voluntary movement on the thirty-first day. Reflex incontinence returned before the knee-jerk, and some control of the sphincters was regained before voluntary power. Sensibility returned in segmental order, and at one period Brown Sequard's symptom was definite. She recovered sufficiently to work, but her gait is slow and spastic (1903).

This case is cited as illustrative of the clinical picture and course of many cases of acute myelitis in this series, details of which are omitted. It is a typical instance of the total physiological lesion, with its classic signs of flaccid palsy and absent deep reflexes, becoming by recovery a partial lesion, with its classic sign of spastic paralysis.

Case 11.—*Pressure paraplegia. Complete motor and sensory paralysis. Knee-jerks retained. Autopsy. Lesion not total.*

E. S., aged 36 years, was admitted into the National Hospital under Dr. Bastian on August 10, 1899. She had had the right knee excised for tubercular disease.

The symptoms of spinal tumour had appeared three months before admission, and had gradually progressed.

On admission there was complete spastic paralysis and complete loss of sensibility below the fifth dorsal level. There was reflex incontinence. The knee-jerks were exaggerated, and the plantar reflexes were extensor. She died on August 18 of general tuberculosis. The pathological examination was made by Dr. Batten. At the level of the fifth and sixth dorsal segments the spinal cord was much compressed and partly invaded by a tubercular growth. Sections of the compressed region stained by Pal's method showed a considerable number of healthy fibres passing through the lesion.

Case 12.—*An incomplete lesion, with absolute and persistent motor and sensory loss.*

A. J. C., aged 26 years, was admitted into the National Hospital under the care of Dr. Bastian on July 5, 1898.

He was seized in December, 1897, with a typical acute transverse myelitis affecting the lower thoracic cord.

There was absolute spastic palsy and absolute anæsthesia to all forms below the eleventh dorsal level. The knee-jerks were exaggerated, and first clonus was present. The plantars were extensor in type. The muscles were not markedly wasted. The sphincter tone was well marked, and there was reflex incontinence, and sometimes retention with dribbling. Priapism occurred till May, 1898, but not subsequently. He died on August 5, 1898.

The pathological examination was made by Dr. Risien Russell, who found that there was no evidence of complete transection, normal medullated fibres being found in numbers in the lateral columns throughout the length of the lesion.

This case demonstrates the fact that the persistence of complete paralysis and complete loss of sensibility cannot be taken as evidence that a lesion is total.

Case 13.—*Pressure paraplegia. Recovery after complete sensory and motor loss, which persisted many weeks. Autopsy.*

M. T., aged 19 years, was admitted at the National Hospital under the care of Dr. Buzzard on June 14, 1898. She had had an angular curvature for twelve years. The paraplegia has appeared five months before admission. There was absolute spastic palsy and absolute loss of all forms of sensibility below the twelfth dorsal level. There was marked relative loss as high as the ninth dorsal level, touch being the least impaired. There was reflex incontinence of the sphincters. The deep reflexes were much exaggerated. She stated that she had been unable to feel anything in the legs for several weeks. Laminectomy was performed by Sir Victor Horsley on July 5, and the pressure was relieved. She made an uninterrupted recovery, and was able to walk well on November 1.

In January, 1899, she developed acute pulmonary tuberculosis and died.

The pathological examination was performed by Dr. Risien Russell. The seat of the lesion was the ninth dorsal segment, where marked signs of old degeneration were visible, as was also evident in the ascending and descending tracts of the cord.

This case is recorded as demonstrating that complete spastic paraplegia and complete sensory loss of many weeks' duration are compatible with complete recovery of the functions of the spinal cord.

Case 14.—*Compression paraplegia. Great local reduction in size of the spinal cord without sensory loss and with some voluntary power. Autopsy.*

F. S., aged 27 years, admitted into the National Hospital under the care of Sir William Gowers on October 1, 1897, suffering with spinal caries. The paraplegia began in May, 1897.

When admitted there was complete spastic paraplegia. No sensory loss and no dysuresis. Treatment by rest and extension for nine weeks were not followed by improvement. In December, 1897, Sir Victor Horsley performed laminectomy. Fourteen days later some voluntary power returned in the toes. He steadily improved, and became able to walk with assistance. Subsequently extensive tubercular disease of the lungs became active, and he died on October 11, 1898.

At the autopsy, which was performed by Dr. Risien Russell, the spinal cord was found to be reduced to less than one-half its normal size for a distance of 7 mm. in the region of the sixth and seventh dorsal segments.

Case 15. — *Total lesion, with implication of lumbar roots. Autopsy.*

W. W., aged 88 years, was admitted into the National Hospital under the care of Sir William Gowers on January 9, 1900.

Symptoms of paraplegia had appeared five months before admission, and had progressed steadily. When admitted, slight voluntary movement of the left lower extremity was possible.

There was no power of movement in the right lower extremity. The legs were spastic. The left knee-jerk was exaggerated, the right was subnormal. There was loss of control over the sphincters.

There was marked analgesia and thermanæsthesia and less marked tactual anæsthesia below the ninth dorsal level. There was slight impairment of pain and temperature sense as high as the sixth dorsal level, while the fifth dorsal area was very markedly hyperalgesic.

On February 20 Sir Victor Horsley performed laminectomy, and found that extensive pachymeningitis was present.

On March 28 the lower extremities were quite flaccid, and the knee-jerks were unobtainable. Loss of sensibility was complete up to the level of the xiphisternum. Repeated attempts were made to elicit the knee-jerks after long-continued faradisation of the legs, but without success. Subsequently the sensory loss spread to the arms, which were involved in atrophic palsy, and he died on March 22, 1901.

At the autopsy extensive hypertrophic pachymeningitis was found extending from the upper part of the cervical enlargement to the cauda equina. There was complete necrosis of the ninth and tenth thoracic segments, presumably from evascularisation. All the lumbar roots were involved in the meningeal growth, and they were all degenerate.

This case is cited as demonstrating the clinical value of the non-return of the knee-jerks upon faradisation of the lower limbs, shortly after the onset of the flaccid state. The sign indicated that the lumbo-sacral enlargement of its nerve roots was implicated, and that the flaccid paraplegia was not solely the result of the total transverse lesion, for in the latter cases the knee-jerks should have been elicitable after faradisation.

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ON THE PRIMARY STAINING OF THE RAT'S BRAIN BY METHYLENE BLUE.

BY JOHN TURNER, M.B.

ONE of the most curious and important features in neurohistology is the extraordinary affinity which methylene blue has for the elements of the nervous system, especially in regard to what Bethe terms its primary staining capacity, *i.e.*, the staining of fresh material, not previously acted on by chemicals, with basic dyes.

By different ways of applying the stain very different results are obtained; thus, at one time it picks out certain details and neglects all others, at another time these details fail to show and quite different ones are revealed.

Ehrlich was the first to apply the stain directly to the tissues, which he did by injecting it during life into an animal. In this case the great bulk of the cytoplasm of the pyramidal cells is scarcely coloured at all, but other cells, scattered irregularly about in the cortex, stain very darkly. There is shown also around some of the pyramidal cells a network of fine, beaded fibrils. My method, which gives a similar, but more detailed picture, enables us to obtain this reaction in dead tissues. This I have called a pseudo-vital reaction, and up to the present time have only succeeded in getting it occasionally, and, as a rule, in small isolated parts of the tissue.

Apàthy, Bethe, Dogiel and others, by a different procedure, succeed in staining the neurofibrillæ of the ganglion cells, but apparently their methods do not differentiate the darkly-stained cells above mentioned.

Under still different conditions the dye seizes on other details in the ganglion cells, and stains the so-called Nissl-bodies (which are probably structures not directly concerned with nerve conduction), and leaves the rest of the cell body uncoloured. Bethe applies the term "primary staining" to tissues which have been dehydrated in alcohol or ether before staining, and therefore regards the colouring of the Nissl-bodies as primary, and Lenhossék and I have shown that perfectly fresh tissues acted on only by the dye clearly show them.

As a rule, in primary staining by methylene blue, it is one or other of the nervous elements which are especially selected. The glia is largely neglected. However, during my attempts to get the pseudo-vital reaction in animals' brains, I have found that occasionally the dye specially picks out certain of the glia cells and structures which, I believe, are also derivatives from these cells.

I propose to give, in the following pages, a short account of these results, and also of some in which I have succeeded in obtaining the pseudo-vital reaction in the brain of the rat.

I shall deal first with the pseudo-vital reaction which I have been able to get in only one or two cases after keeping the dead animal for twenty-four hours in the oven, at 25° to 30° C., or else by taking out its brain and leaving this organ under similar conditions in a closed glass vessel. The staining mixture, consisting of 40 cc. of 1 per cent. methylene blue, to which 2 drops of lactic acid has been added, and 4 cc. of peroxide of hydrogen (10 vols.) was slightly warmed, and the brain put into it in a covered vessel, and left in the oven (25° to 30° C.) for six or seven days, then fixed for twelve to twenty-four hours in 10 per cent. molybdate of ammonium, washed, dehydrated, embedded in paraffin and cut. The successful results having been limited to very small areas in the cortex and striate body of two or three brains, I purposely give a very cursory account of the procedure, as I am still experimenting, and hope before long to be able to arrive at more constant and more widespread results. After a great number of trials it is evident (1) that the reaction does not occur in perfectly fresh tissue; (2) that the addition of a trace of lactic acid to the staining fluid somewhat facilitates matters (with this observation we must connect the fact that lactic acid is developed in dead nervous matter); (3) any signs of putrefaction due to aerobic organisms prevent a successful result, and at the same time, by setting free ammonia compounds, give an alkaline reaction to the tissue.

During these investigations I found if the brain was subjected to a certain heat, when immersed in the staining fluid, that a very different result from that sought after was obtained. Scattered irregularly throughout the cortex and white matter were small, multi-branched cells which stained absolutely black, and which appeared to be glial. Furthermore the apices of the pyramidal cells frequently stained deeply and showed numerous fine, short, lateral projections, ending in knobs; and, lastly, around many of the nerve-cells beautiful Golgi nets were shown.

(*A*) PSEUDO-VITAL REACTION.

Cortex cerebri (median aspect adjacent to the corpus callosum).—The cytoplasm of the pyramidal cells (including the cells of the second layer) and the spindle-cell layer, stain a pale sky-blue and quite homogeneously; the nucleus is somewhat darker and densely granular; except the apex, the other protoplasmic processes are practically not shown. Scattered here and there throughout *all* layers of the cortex are small (an average size being $14 \times 9\ \mu$) multi-branched cells, not definitely orientated, staining nearly black and giving off extremely long, delicate processes, which spring abruptly from the cell body and pursue a very sinuous course. These processes are not regular in contour, but present a number of fine beads or more elongated thickenings, lying close together connected by finer strands, and with high magnification the thicker parts do not appear quite homogeneous, but have a loose structure, something like cotton wool, and in favourable cases they show numerous delicate, lateral twigs, similar to those on the processes of certain of the striate body cells. The processes ramify freely within the first layer (fig. 1). The pericellular beaded network is very scanty; indeed, scarcely more pronounced over the cells than in the intermediate regions. Its fibrils are extremely fine and the black beads are generally about 2 μ apart, but the distance between them varies considerably as in human brains. So far I have not seen anything approaching the dense fibrillar felt work such as exists between the cells in the human cortex. The fibrils forming the pericellular net work appear very similar to the finest branches of the dark cells: in both cases the structures are beaded, but usually the dendrites appear less homogeneous and sharply cut than the fibrils of the network.

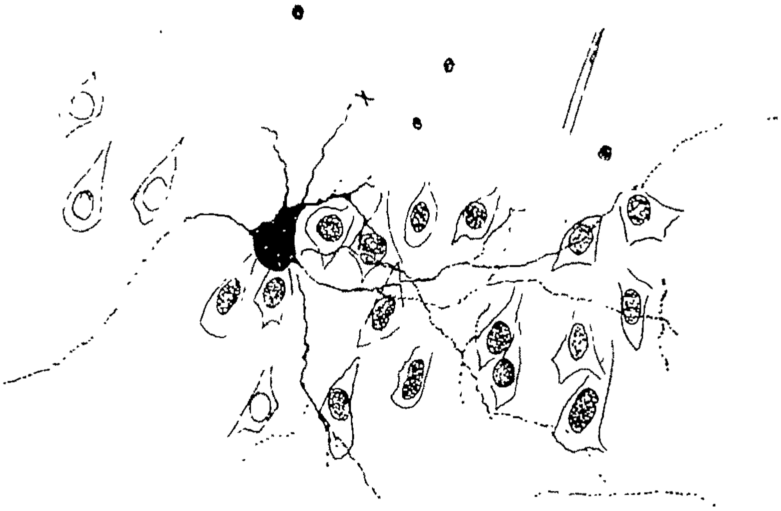

FIG. 1 (× about 500 diameter).

Small, darkly-stained cell in the second layer of the cortex. Its branches are extremely fine, and pursue a sinuous course for very long distances, retaining the same calibre. Note how they spring abruptly from the cell-body, and that some of them pass up into the first layer. X probably represents its axon passing up into the first layer.

Contrary to my universal experience with human brains, the first layer in rats, I find, takes on the reaction and shows considerable fine structural details. Fairly numerous extremely fine black fibrils run in it at all depths parallel to the surface. They have a clean-cut aspect and present at irregular intervals minute swellings, sometimes beads. Similar fibrils pass up into this layer from beneath, and bend over to one side or the other. A fair number of nerve-cells are met with, staining the same light-blue colour as the cells in the lower layers. Occasionally also here and there the apices of pyramidal cells which have stained black are seen traversing this layer and bifurcating in it; sometimes they reach nearly to its outer margin, and it is to be noted that along their sides are closely clustered black dots, which join to the dark process by a delicate pedicle. In the few cases where I have been able to trace these dark apices to their parent cell the lateral offshoots were not apparent over the cell body, and only began to show at some little distance from it on the processes. These side twigs have very much the appearance of those on the dendrites of the striate body cells, but are shorter and more thickly clustered.

Cornu ammonis.—My remarks refer to the long pyramidal cells and the stratum radiatum. Only the bodies of the pyramids show, and they stain of an uniform pale blue, their nucleus not much darker. The ground work of the stratum radiatum and the spaces between the cells, where they are not so closely packed together, stain a dull pale gray, and coursing in the groundwork, usually in a direction more or less direct from the bodies of the pyramids to the granule layer, are a number of very fine, dark processes which originate from darkly-stained cells lying here and there among the pyramids or in their immediate neighbourhood. These dark cells are similar to those already described, but are, as a rule, somewhat larger (18 μ), and often more or less bipolar.

In the places where the bodies of the pyramids lie closely packed together they are marked off from each other by a darkly-stained beaded line, which, in places, gives off branches which pass over the cell bodies. Where the pyramidal cells do not lie quite so close together the fibrils course sparingly throughout the intermediate substance (see fig. 2).

Striate body.—This shows a structure very similar to the human caudate nucleus. Its cells are nowhere definitely orientated; the pale variety, much the more numerous, are chiefly indicated by their nuclei, as the cytoplasm is only faintly coloured just around these bodies. A few angular cells stain deeply and give off comparatively short rectilinear processes, which dwindle rapidly and pass from view, and these I am inclined to regard as cells belonging to the pale

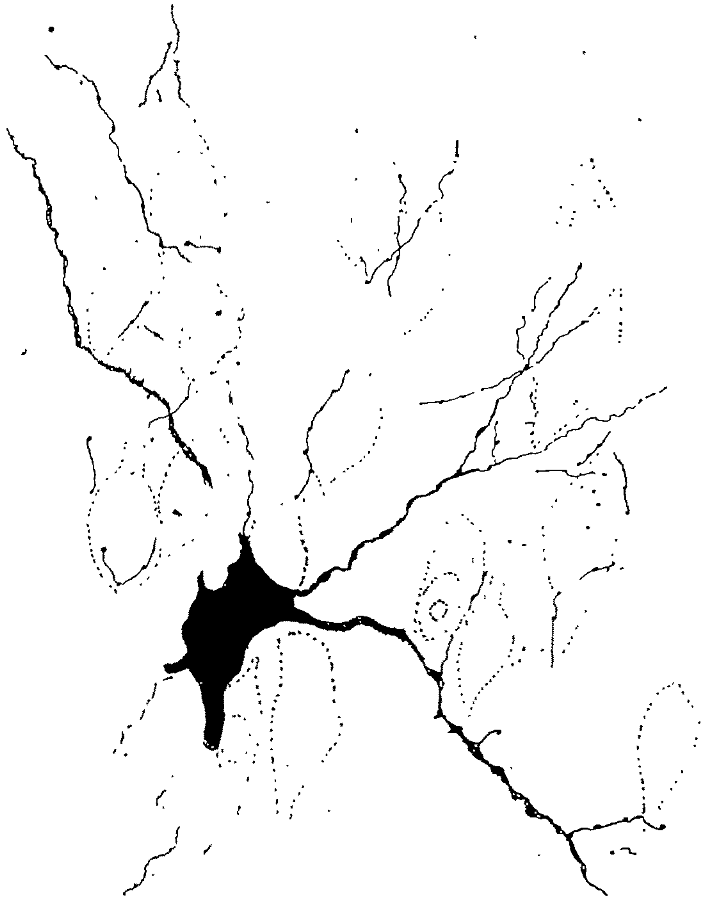

FIG. 2 (× 780)

Darkly-stained cell, lying in amongst the long pyramidal cells of the cornu ammonis (which are represented in outline only). Fine, beaded fibrils invest the pyramidal cells sparsely, and in some cases appear to take origin from the dendrites of the dark cell.

category, which have, for some unknown reason, coloured darkly.

The dark cells are exactly like those I described and figured in the human caudate nucleus (Brain, Autumn, 1903). Their long, slender, sinuous processes form a coarse felt-work with each other, and generally in their distal parts

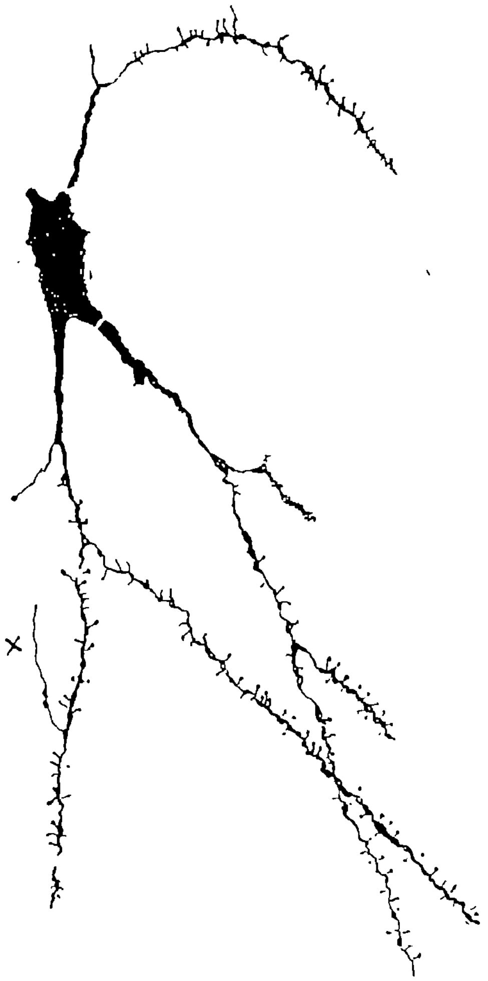

Fig. 3 (× 780).

A dark cell from the striate body. Delicate lateral twigs ending in a bead are given off from the more distal portions of the dendrites. At X a long, beaded fibril appears to arise from a dendrite.

they give off numerous small, but distinct, lateral twigs ending in a knob a little distance from their origin, or else long fibrils with small heads on them at short intervals (fig. 3). Not all the dark cells show these "thorns"; the processes of some present a very irregular, moniliform, or coarsely-beaded appearance.

Numerous bundles of fine axons traverse this body on their way to the cortex, and where individual fibres can be detected they are of a pinkish hue and do not show definite beads, although their contour may not be perfectly smooth.

REMARKS.

Although the results so far obtained are meagre, they show that the "dark" cells occur in both the first and second layers of the cortex and in the cornu ammonis, in which places I have not yet succeeded in showing them in the human brain. The presence in the first layer of the dendrites of these cells, especially of those cells which lie just below it, and which send up processes to ramify therein (*vide* fig. 1), might be supposed to militate against the idea that the beaded pericellular network is a derivative from these dendrites, because this layer is poor in cells. But in the new-born infant and in full-grown cats, as I have frequently observed, it does contain a considerable number of cells, the chromophilic elements of which are fairly well marked, and arranged in a similar manner to those in the pyramidal cells lying lower down in the cortex, a characteristic which at all events appears to mark them off as distinct from the dark cells. Very likely also in rats' brains these chromophilic cells are persistent throughout life, as in the cat.

I believe that not only do these dark cells form an intermediary system between the end afferent stations and the first efferent (*e.g.*, in the cortex), but that they are met with at afferent stations of different levels (*e.g.*, Clarke's column nuclei, gracile and cuneate nuclei, optic thalamus, &c.), interposed between the terminals of one level and the cells of the next; and that in all probability they will be met with similarly disposed at the efferent stations of different levels, both cerebral and cerebellar.

Golgi long ago described cells to which he gave the name of intercalary; very probably these are included with others (notably Martinotti's cells) in my dark-cell system, and therefore I propose to call it the intercalary nerve-cell system.

Besides their staining peculiarities they have other well-marked characteristics by which they can be identified, and which, I submit, afford reasonable grounds for regarding them, wherever situated, as of like nature.

These are four in number, viz.:—

(1) *Dark staining.*—This, although a useful character by which their presence is readily revealed, is not alone to be depended on, for although, so far as I am aware, they always stain deeply, yet occasionally cells of the pyramidal system take on a dark colour also, and as this occurs very often in the brains of presumably healthy rats, especially in the cornu ammonis, it probably does not necessarily depend on a pathological condition, but possibly on some chemical alteration accompanying the functional activity of these cells.

(2) *Shape.*—Although in this respect they vary very much, yet they are extremely rarely pyramidal. They are usually square, pentagonal or oval, but whatever form they assume their protoplasmic processes spring from them abruptly and do not pass off by insensible degrees as, *e.g.*, the apical process of a pyramidal cell; sometimes, especially in the oval forms, a thick process springs from the cell in such a manner that one is unable to determine exactly where it leaves the cell body, but in such cases, after a very short course, it divides into two much smaller branches, which run for very long distances without alteration in calibre.

(3) *Dendrites.*—The way in which they arise has just been described. They run for very long distances without marked diminution in calibre, and are irregular in outline, with either moniliform swellings or lateral twigs. Their course is sinuous, and not, as is almost universally the case with dendrites of the pyramidal cells, rectilinear. This last feature I look upon as very characteristic.

(4) *Orientation.*—Unlike the pyramidal cells they are never definitely orientated.

In my previous communications I have given instances in the human brain which seem to demonstrate a direct connection between the protoplasmic processes of the intercalary cells and the beaded network around the pyramidal. In dealing, however, with such extremely minute structures, dogmatic statements must be received with caution; but, apart from such seemingly direct evidence, there is much of an indirect nature which also favours this assumption. I would especially call attention to the long, beaded fibrils which pass off from the dendrites of the intercalary cells of the striate body, and which have identically the same appearance as those forming the pericellular network. The objection that from this point of view the so-called law of dynamic polarisation is contravened, has with our present knowledge of the intimate structure of nerve-cells no longer much, if any, weight. The presence of neurofibrillæ, and the way in which they are arranged in the cells, shows that, as applied to the pyramidal cells, this doctrine is untenable. It is now a well-established fact, which anyone can verify for himself, that neurofibrillæ pass directly from one dendrite to another, and therefore, in whichever direction the nervous impulse traverses such a fibril, it must pass in opposite directions in the two dendrites in which a particular fibril lies. I am assuming that there are such phenomena as nervous impulses, and that they pass along neurofibrillæ. For the experiments of Bethe seem to show that the fibrils are the only material substance along which they can pass, for, according to him, they are the only structures which run continuously through the nodes of Ranvier.

(*B*) HEAT-FIXED REACTION.

If the staining mixture is added to the brain and cord and the whole subjected to heat in a covered glass capsule for some time, and then left in the oven at 25° to 30° C. for three or four days, one obtains occasionally, especially in young rats, a quite different reaction. I am unable to give any hard and fast rules, as my experience is not sufficiently extended, but I have obtained successful results in several cases, more especially when the brain in its staining fluid was slowly raised to 60° C. on the water bath and then allowed to cool to 50° C. and again slowly raised to 70° C. and kept at this last temperature for some time, according to the size of the material to be stained (half to one or more hours). It is then placed in the warm oven (26° to 30° C.) for four days, and the subsequent treatment is as before described. In one case, which showed the lateral projections on the processes of the pyramidal cells, the brain had been subjected to a temperature of 100° C. for several minutes; in another, which showed Golgi nets around the Purkinje cells, the temperature was only raised to 50° C. and kept at this point for two hours. Under these circumstances, with successful preparations, one gets appearances quite distinct from the pseudo-vital reaction. No darkly-stained, intercalary cells, nor beaded network, nor other fine detail of an undoubted nervous nature is shown, but numerous glia cells are shown, sparsely scattered about in the cortex white matter, and in in the central nuclei and stem ; and besides these well-developed cells with many branches, a number of round or oval bodies are seen, which vary in colour from a dull pink to almost black. They are about 9 μ in diameter and generally have a pale, round area within them occupying the greater part of their mass. These bodies are especially numerous in the white matter and amongst the cells of the nuclei of the stem. A variety of transitional forms between these just described and fully developed black glia cells occur ; at one end of the scale there is an oval mass without processes ; one passes from this to intermediate forms which show several short unbranched processes; at the other end of the scale the processes have become long, numerous, and freely branched, and the body less round (see fig 4). I have observed all forms of these cells in large numbers in the human cerebrum and cerebellum, treated in the manner first described for the rat's brain (*i.e.*, raised to 70° C.). In the latter region, cells which appear quite similar to the immature-looking forms in the rat's brain make up a large bulk of the entire granular layer (see

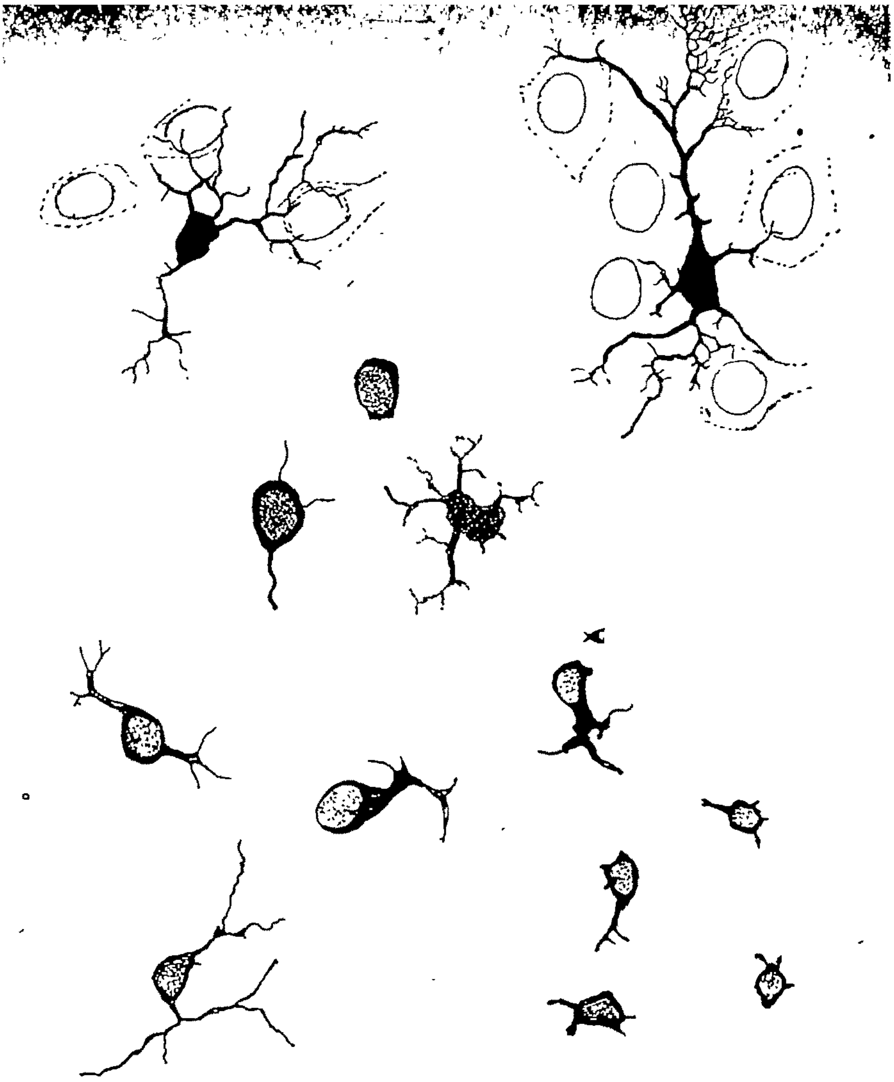

FIG. 4 (× 780).

In the upper part are two well-developed multi-branched glia cells lying in between the bodies of the long pyramids of the cornu ammonis. They are much smaller than the dark nerve-cells which are met with in this region (*vide* fig. 2). In the right-hand figure the end-branchings are shown (somewhat diagrammatically), breaking up into a lattice-work covering for the pyramidal cells.

In the middle of the page are three glia cells from the white matter, showing more or less immature-looking forms; the upper has no processes visible, the left-hand one has three stunted, unbranched processes, and the right-hand one has many. Note the pale area within two of these bodies.

In the lower part, to the left, are three well-developed glia cells from the molecular layer of human cerebellum. To the right are four small glia cells from the granular layer, with stunted branches. These are often clustered together into clumps of three or four. At **X** is a somewhat longer (8 by 5 μ) and more developed cell, which lies in the lowermost part of the molecular layer, and has the appearance of being a more developed form of the granular glia cell. (The pale centre, although represented as granular for facilities of reproduction, is really quite homogeneous).

right hand side of fig. 4, lower half). At the junction between this and the molecular layer and in the innermost part of the latter the cells become larger and more branched, whilst in the outer parts of the molecular layer well-developed forms occur (left hand fig. 4, lower half). In nearly all cases they show a pale central part.

My sections show very clearly that there is a very intimate relation between the branches of these cells and the trabeculæ of the Golgi nets; in fact, I believe they demonstrate a direct passage over of their branches into the net structure.

Golgi nets.—These are shown very distinctly, closely investing the bodies and dendrites of the cells of the dentate nucleus of the cerebellum, and most of the cells of the different nuclei within the pons, especially those of Deiters' nucleus, and the fore horn cells of the cord; they appear less distinctly shown around the pyramidal cells of the cerebrum, the cells of the corpora quadrigemina and the Purkinje cells.

They consist essentially of a close-fitting trellis structure, something similar in appearance to a piece of perforated zinc; the apertures are usually polygonal, and often contain a dark dot in the centre; the trabeculæ are slender. Bethe states that cells of different classes have nets of different patterns, so that from inspection of only a small piece of the net one is able to tell whether it belongs to a cell from the cortex, dentate nucleus, or trapezoid body, &c. The meshes over the dendrites are usually more elongated than those over the body. This authority also states that the net invests the axon for a short distance, ceasing either before or at the place where the myeline sheath begins.

It is very evident to the most casual inspection that this investment and the beaded pericellular network revealed by the pseudo-vital method are entirely distinct.

I have not been able to make out any extension of the trabeculæ of the Golgi nets to the matrix between the cells such as Bethe describes, but where two cells lie in apposition it passes continuously from one to the other, as he also notes. My preparations appear to show very conclusively that they are derived from the terminal ramifications of the branches of glia cells, and although my personal acquaintance with these nets is confined to sections from several rats' and human brains, yet I have already met with, in the former, many instances which appear to establish this connection without doubt (see fig. 5).

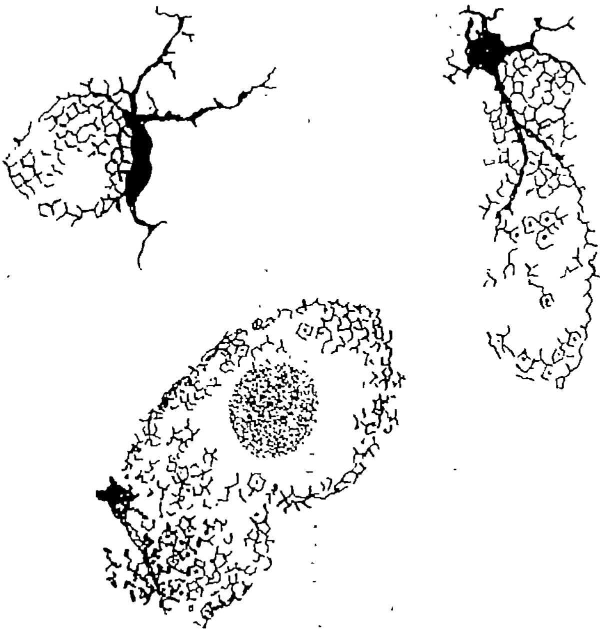

FIG. 5 (× 780).

Golgi nets surrounding, in the upper part of the figure, two cells from the dentate nucleus of the rat's cerebellum. In the lower part a large cell from one of the nuclei of the stem. In each case the darkly-stained glia cell from which the net appears to arise is shown. Note the pentagonal form of the mesh-work, and the frequent presence of a black spot in the centre. In the two upper figures the nerve-cell is merely indicated by its covering, but in the lower one the nucleus is shown.

Thorns.—In these heat-fixed preparations very often the apices and main dendrites of the pyramidal cells of the cortex stain somewhat darkly. The apices, under these circumstances, can be seen throughout almost the entire depth of the cortex, like a forest of pine trunks, lying closely together and ending apparently in the inner part of the first layer, where they incline slightly to one side or the other; sometimes, especially in the case of the second layer cells, they bifurcate. The bundles formed by these apices are reinforced as they pass up by apices from shorter cells, so that they are more densely packed towards the surface than lower down. The cell body itself does not stain deeply, but the dark colour begins somewhat abruptly not far from it. The axons of these cells also stain deeply, but dwindle and disappear after a short course. In the more distal parts of the apical process, very fine, closely set, short twigs, ending in a knob, are seen passing away at right angles from its sides, and in cross sections through the dark apices these projections can be seen passing off all round like the spokes of a wheel (fig. 6). They appear quite similar to the lateral twigs already referred to, which show when the pyramidal apices stain darkly by the pseudo-vital method. Very often, also, in this view, the cut-through process is only dark on the surface and has an almost colourless centre. When examined under a high power it is seen that the dark colour is not uniform, but consists of darker granules on a somewhat lighter background. The beads can often be distinctly seen joined to the dark apex by delicate pedicles, but very often, in the immediate neighbourhood, others are seen scattered fairly thickly about and showing no connecting fibrils.

REMARKS.

Golgi nets were originally described by the distinguished Italian histologist of that name in 1882. He ascribes to them an isolating function, and believes that they are a neurokeratin structure, but opposed to this idea is the fact, which Bethe points out, that this substance is not digested by pepsin, whilst the network is. Ramon y Cajal, Donaggio, Apàthy, and, recently, Held, regard them as glial in nature, and the two first as representing merely a peripheral extension continuous with the endocellular network of Golgi. Bethe and Semi Meyer believe that they are nervous structures, and Bethe represents them as extensions of axis

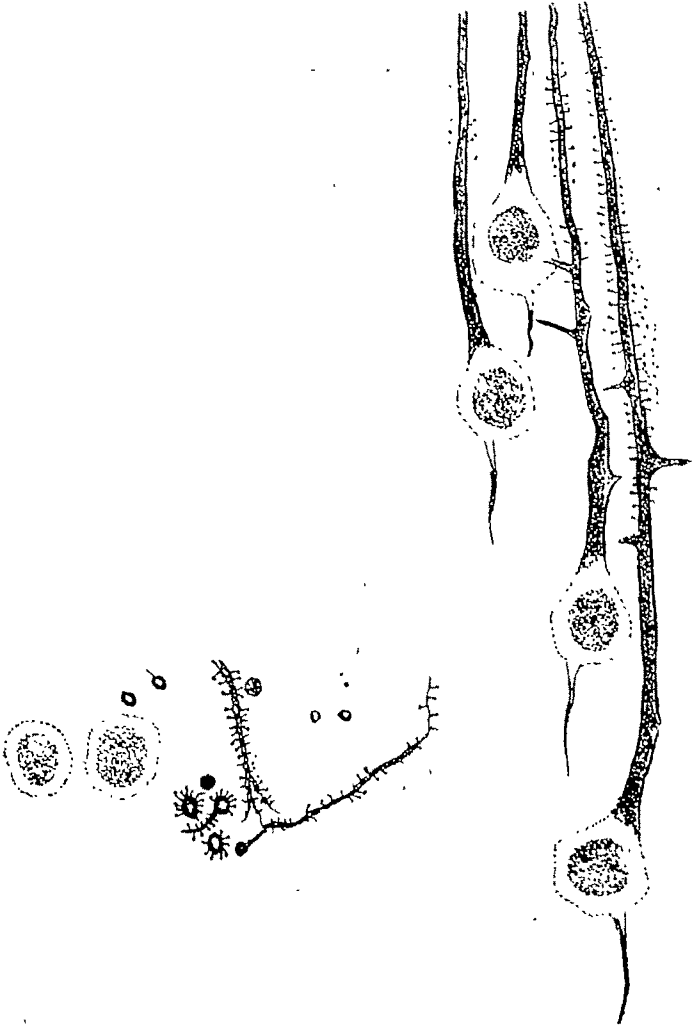

FIG. 6 (× 780).

On the right side are four pyramidal cells with darkly-stained apices. At some distance from the cell body fine dots are visible at the side of these processes, and generally a fibril connects the dot to the edge of the process. But, in places, collections of these dots occur which do not seem to have any connection to the pyramidal cells, as shown on the right side of the longest cell.

The left-hand figure gives a cross section of the pyramidal cells.

The apices now appear as a black spot or a black ring with colourless centre, and the little lateral projections come off all round like the spokes of a wheel. The long processes with thorns are dendrites. The two large, pale nuclei, with clear rims, represent two pyramidal cells cut through their bodies. Although these figures were outlined by aid of Zeiss's Camera Lucida, the lateral projections are so minute that it is not pretended that they have been individually traced, but only give the general appearance produced to the eye.

cylinder processes which, coming from other parts, join to the network and are thus brought into relation with the enclosed nerve-cell. The neurofibrillæ contained in the axis cylinders pass along in the trabeculæ of the nets, and leave them to enter the substance of the ganglion cells within. He believes that these nets, and a diffuse network which in places he sees in connection with them lying in the general ground substance, are identical with Nissl's hypothetical "gray" (but on this particular point Nissl does not agree with him)

My observations point strongly to the glial origin of these structures. The fact that I have only succeeded in showing them in preparations in which the stain only selected elements which appeared to be of a glial nature favours this view, but apart from such indirect evidence I have, as already mentioned, frequently seen what has every appearance of being a direct continuity between the branches of the glia cells and the network.

It is a pity that there are not greater facilities whereby different workers could compare one another's sections; if this were feasible probably many discrepancies and seemingly antagonistic views would be brought into harmony. This is the more to be desired as modern methods of staining nervous structures tend to become more special and limited in their range, so that taking any one method, whilst some details are shown, others which, perhaps, may be very closely related, are not revealed at all; and although one worker may not, and usually does not, limit himself to a single method, he will probably not reach an equal degree of perfection at all; thus, whilst he may obtain results which, after a careful examination of very many sections, appear to him convincing, he has not the time, nor perhaps the opportunities, to examine with equal thoroughness large numbers of preparations stained by different methods. Taking the case of these Golgi nets, I feel very strongly that my sections do show that they result from the splitting up of the glia cell branches, but I am unable to speak of their relation to the endocellular network, because the method I use does not show the slightest trace of these structures. If, as Cajal and Donaggio think, they are both parts of one and the same thing, and in view of Holmgren's observations on the intracellular networks and "sap" canals in posterior spinal ganglia and gland cells, then they are probably a mechanism concerned with the metabolism of the cell.

On the other hand, the junction of axons with Golgi nets is strongly insisted on by Semi Meyer, and Nissl, after an inspection of Bethe's specimens, was also convinced of it. Nevertheless, in spite of the weight which opinions from such eminent observers carry, the minuteness of the structures under discussion must make it extremely difficult for anyone to definitely decide between actual continuity or mere proximity. It may be that the axons surround the ganglion cells, enter at the small pentagonal openings of the net, and break up within into fibrillæ. But, even should Bethe's observation prove correct, I should still be inclined to regard the network as not essentially a nervous structure, but as affording a covering for the fibrillæ, which Bethe states he has seen *within* its trabeculæ, without being itself concerned directly with the functional nervous activity of the ganglion cell.

On the assumption that the Golgi net is an extension of the axis cylinders rests the main fabric of Bethe's argument for the continuity throughout the central nervous system of individual neurofibrillæ as expounded by him in his recent publication, "Allgemeine Anatomie und Physiologie des Nervensystems," and this, to my way of thinking, is a weak link in his chain of reasoning. I myself believe in the continuity of nerve-cells with each other by their processes, but I think it occurs in a different way to that which he describes.

Thorns.—The thickly-clustered, delicate, lateral offshoots from the darkly-stained apices and dendrites of the pyramidal cells are to be seen, as already mentioned, not only in heat-fixed preparations, which do not, apparently, lend themselves to the staining of fine nerve details, but occasionally on the pyramidal apices which have, contrary to their usual custom, stained darkly in preparations which give the pseudo-vital reaction. Although they show a great resemblance to the projections on the dendrites of some of the darkly-stained cells in the striate body, I am inclined to think there is a distinction between them and those on the pyramidal processes, and that the latter are not integral parts of the cells to which they adhere. They appear to me to be finer, more closely clustered and shorter than those on the striate body cells. Without accepting the interpretation which Bethe gives to the function of the Golgi nets, perhaps, as he suggests, these lateral projections represent the stained trabeculæ of Golgi nets passing from the cellular to the diffuse portions of these structures. An objection to this view is that whilst we know that the Golgi net envelops not only the processes but the cell body, this latter part does not show the lateral projections, either with the pseudo-vital or heat-fixed methods. Speaking now of the appearances seen in heat-fixed preparations, if the Golgi net over the apices causes them to stain deeply, we may reasonably ask why the cell body is not also similarly affected. This may possibly be due to a difference in the size of the meshes over the cell and processes; if those over the dendrites are smaller they might hold the stain so as to cause the whole surface to appear dark, whilst the bigger meshes over the cell body would fail to do so.

However this may be, I do not feel qualified at present to express any decided opinion as to their precise nature, except to reiterate what I have before stated, that they do not appear to me (especially in the heat-fixed preparations) to be integral parts of the processes themselves.

A few words in conclusion concerning the differentiation by the heat-fixed method of the granules of the human cerebellum. There is still much uncertainty about the nature of these bodies; true it is that Golgi's method shows here and there in the granular layer a small cell with ascending axon and claw-like dendrites, but these only occur in comparatively few numbers, and I have always felt that their presence was no sufficient justification for looking on all the granules as of the same nature. My preparations show that a very large number stain differently from the rest. These stain deeply, show a few stunted processes and a pale central portion. They resemble very closely the immature-looking glia bodies in the rat's brain, and show grades differing only slightly one from another between the most immature form and the well-developed glia cell, and to my mind can with very little doubt be classified as glia elements. But as I have, up to the present, only seen these bodies in more or less pathological brains, I merely wish to draw attention to the fact without drawing any conclusions therefrom.

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All the figures were drawn by the aid of Zeiss's Camera Lucida.

NOTE ON A CASE OF DEFECTIVE DEVELOPMENT OF THE LATERAL CEREBELLAR LOBES IN A DOG.

BY THOMAS LEWIS.

(From the Laboratory of Chemical Pathology of University College, London.)

THE interest attached to the present case of cerebellar defect lies in the fact that it affords suggestions concerning the physiology of "circus movement" in an animal in whom the cerebral hemispheres were normal. Circus movement (*mouvement de manège*) occurs most commonly in animals in cases of lesions of the cerebral hemispheres producing hemianopia.

Luciani[1] has described the path taken by a dog in walking, after unilateral ablation of the cerebellum, as festooned to the opposite side, while the course followed by the same animal in swimming is stated to have been curved to the *same* side as the lesion.

In regard to the ponto-spino-cerebellar mechanism there are at present generally recognised two main groups of movements, the first included under the term *mouvement de manège*, the second comprising rotatory movements around the longitudinal or transverse axis. In the case at present under consideration *mouvement de manège* was alone present, the diameter of the circle of movement being little more than the length of the longitudinal axis of the animal. Further, it is to be noted that in thus speaking of circus movement and in attributing it to a defect in the cerebellum, it is not suggested that it is strictly comparable to the large circular movements which Prof. F. O. Guldberg[2] has shown to be phylogenetically so important, and (in conjunction with Prof. C. Guldberg[1]) proved to be associated with asymmetrical development of the two halves of the body.

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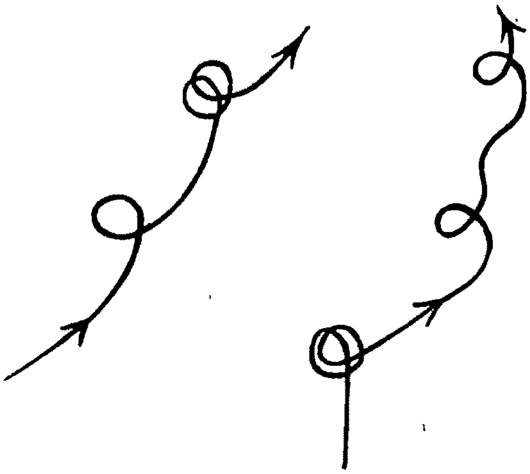
[1] *Il Cervelletto.* "Nuovi studi di Fisiologia normale e patologica," 1891.

[2] *Zeitschrift für Biologie*, T. xxxv.

HISTORY OF THE DOG.

The dog, a collie, was described by its former owner as probably between 6 and 7 years old. It had been in his possession for four years; during the whole of this time the animal had rendered itself conspicuous by its curious gait and excitable disposition. It was in the habit of prowling up and down the road, executing at more or less short intervals complete revolutions to the left. The movements were more frequent and rapid at such times as were likely to excite the animal—for instance, at the passing of vehicles and strangers. The condition was said to have become slightly exaggerated during the three months previous to its having come under observation. The state of the dog and its habits were watched for several weeks prior to its death, and the reported nature of the movements confirmed. The circles described by the dog were of very constant diameter, being between 1 and 1½ yards. Two tracings of typical paths taken by the dog in trotting show the frequency and direction of the



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[1] "Etudes sur la dyssymetrie morphologique," Christiana, 1897.

circus movement during excitement. The dog's posture in lying, while under observation, was invariably on the side, with the trunk bent so that it was concave to the left. When the animal was unexcited progression was practically normal. Examination of the senses of sight and hearing showed no defect in the function of eye or ear. The reflexes in the lower limbs were equal on the two sides and normal.

These clinical observations being verified it was decided to test the corresponding cerebral mechanism by electrical stimulation of the cortex. The animal was placed under an anæsthetic and the following experiment was performed by Sir V. Horsley. The rectus femoris on each side was attached to a spring myograph, and the leg area on each side stimulated alternately. No irregularity of response was detected between the two sides. Further, excitation of the lateral lobes of the cerebellum was also without appreciable effect on the spinal reflexes. The animal was then killed and the brain and cord subsequently removed and examined.

MACROSCOPIC ANATOMY.

Cerebrum.—The hemisphere appeared to be perfectly normal and symmetrical.

Cerebellum.—The chief features of interest were:—(1) a general diminution in size relative to the cerebrum; (2) an increased width of the paramedian sulci, and greater depth of the fossæ on either side of the vermis; (3) a general shrunken aspect of the dorsal surfaces of the lateral lobes.

Pons and Medulla.

The basal aspect of the cerebellar tracts is shown in fig. 1, to which reference should be made.

Superior peduncles.—These tracts showed a slight general diminution in size.

Middle peduncles.—These showed no variation from the normal.

Inferior peduncles.—The *dorsal* cerebello-spinal tract, as it turned up to form the supeficial layer of fibres of the resti-

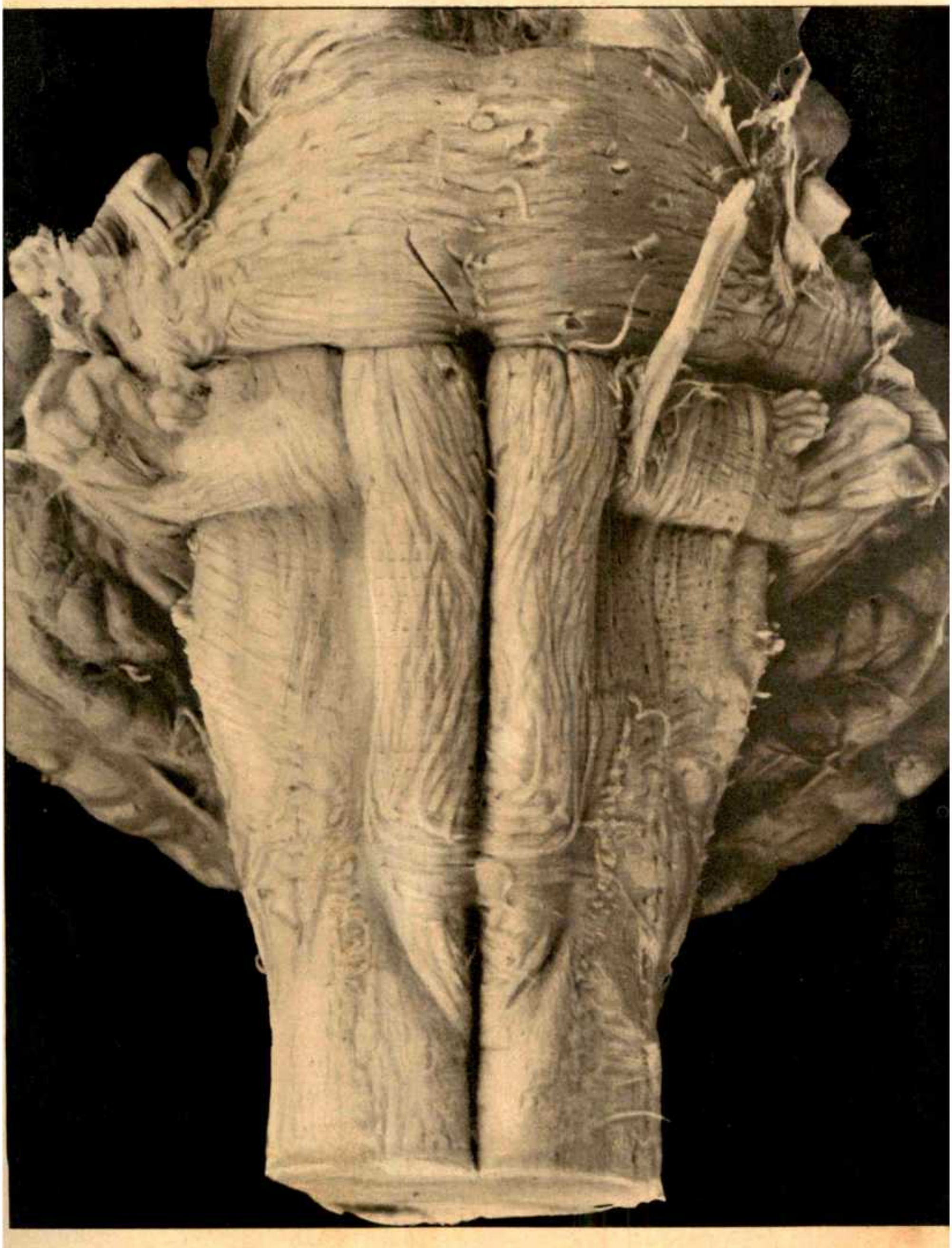

FIG. 1.—Anterior aspect of bulb, showing the ventro-lateral and arcuate fibres. Magnified 5 diam.

form body, was spread out in a thinner sheet than usual. The *ventral* cerebello-spinal tract similarly presented the appearance of more scanty and more widely separated bundles than is customarily found.

Olivary bodies.—These structures were diminished in size and the olivary fibres were poorly developed.

Cranial Nerves.

The cranial nerves showed no abnormalities in size or in origin.

MICROSCOPIC ANATOMY.

Cerebrum.—The cerebral hemispheres gave no indication of morbid changes of any kind, either in the general arrangement or appearance of the cells or fibres connecting them. The optic thalamus and corpus striatum on each side were also normal.

Cerebellum.

The vermis.—In studying the cerebellum by an examination of serial sections the first point of striking import was that the vermis throughout was practically normal. In a paper of Dr. Risien Russell,[1] on the cerebellum of a puppy, to be alluded to directly, the same point appears very clearly, namely, that the mesial plane, the vermis, the phylogenetically oldest part of the cerebellum, shows the least change in these cases of defective development.

The lateral lobes.—The folia were but poorly developed. The molecular and granular layers showed no alterations in depth or in irregularity, such as were found in the puppy already referred to, and in the two cases reported by Herringham and Andrews.[2] The condition of the cells of Purkinje constituted the chief interest in the present case: they were less numerous than usual over the greater part of the lateral lobes; but more particularly was this the case in the folia on the dorsal or posterior, as opposed to the ventral or anterior, surface. In some places they were simply more diffusely scattered, in other situations they were totally absent.

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[1] BRAIN, 1895.

[2] *St. Bartholomew's Hospital Reports*, vol. xxiv.

These points are clearly shown in the accompanying fig. 2.

The flocculus on either side was histologically normal.

The central cerebellar nuclei appeared to be, both in general outline and in the number of cells contained, of normal structure.

Deiters' nuclei.—There was no abnormality in these nuclei, nor in the tracts connected with them.

Pontine cells.—No departures from the usual distribution were detected.

Medullary tracts.—Excepting the dorsal and ventral cerebello-spinal columns, these were of normal appearance.

Spinal cord.—The sole abnormality discovered in the spinal cord was in the central canal, which was throughout considerably dilated, and presented the triradiate calibre of the embryonic type.

CONCLUSION.

It will be seen from the foregoing account that the present case clearly suggests a topographical representation of "circus movement," viz., that this abnormality of progression is associated with the lateral lobes (and so with the pontine nuclei) alone.

The case thus lends weight to the view that the cerebellum may virtually be regarded as a twofold organ, consisting of one part, the vermis, essentially spino-cerebellar, and another part, the lateral lobes, essentially ponto-cerebellar.

I take this opportunity of expressing my gratitude to Sir Victor Horsley for the generous and valuable assistance he has given me. I am also indebted to him for the figures.

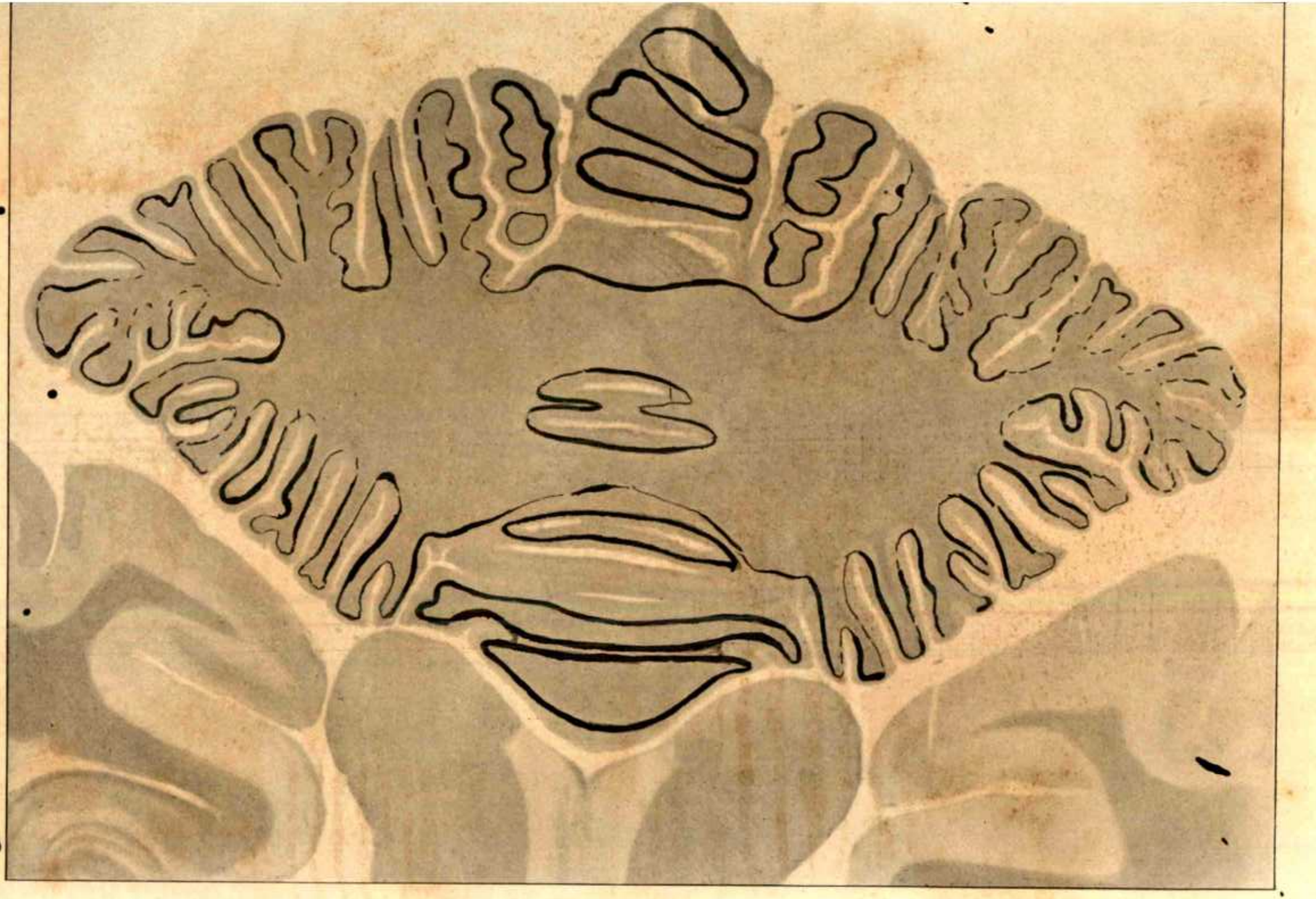

FIG. 2.—The black line indicates by its thickness the number relatively of Purkinje cells in the cortex cerebelli.

UNILATERAL CONGENITAL LESION OF MEDULLA AND SPINAL CORD; DEATH FROM PONTINE HÆMORRHAGE.

BY PURVES STEWART, M.A., M.D., M.R.C.P.

Assistant Physician to the Westminster Hospital.

A HOT-POTATO hawker, aged 41, came to the out-patient department of the Westminster Hospital on account of chronic nephritis, the urine being moderately albuminous and with a few granular casts. It was observed that he had marked asymmetry of the muscles of the neck. He was admitted as an in-patient, and on further investigation the following history was obtained:—

No information can be had about the patient's father. He himself is the fourth of six children. Before his birth his mother, during pregnancy, is stated to have had a severe fall in the street, but at what period of the pregnancy this occurred is not known. One sister, aged 40, is deaf and dumb, one brother is alive and well, three other sisters are dead, cause unknown. The patient as a child was slow in learning to talk, nor was he able to walk alone until 4 years of age. In the lower jaw on the right side he has never had any molar teeth, either of the first or second dentition. All the other teeth erupted normally. The right side of the neck has always been noticed to be peculiar in outline.

Many years ago he had gonorrhœa, and still possesses a urethral stricture. He has been subject to chronic bronchitis for many winters.

On examination he is found to be a man of only moderate intelligence. Speech and articulation are normal, but the voice is hoarse. Smell and taste are normal on both sides. Hearing is somewhat dull in both ears, especially on the right side. The right auditory meatus is narrower than the left, and also higher up. There is no otorrhœa. The right pinna is much smaller than the left, its lobule being particularly diminutive. The right mastoid process is absent (see fig. 1).

The optic discs are healthy, vision is acute, the pupils are equal and normal in their reactions, and the external ocular muscles are normal. The masseter and temporal muscles are strong on both sides, but, on opening the jaw, it swings obliquely to the right, owing to deformity of the mandible, which is smaller on that side (see fig. 2). All the molar teeth of the right lower jaw are absent, the canines and incisors being present. The teeth of the left lower jaw and of both upper jaws are normal in number and in development.

The facial muscles, upper and lower, are normal on both sides, the platysmæ standing out strongly on bending the head forwards.

The right arch of the palate, at rest, stands nearer to the middle line than the left, and, on phonation, the left side moves alone, pulling the raphé upwards and to the left (see fig. 4).

The right sternomastoid is absent. So also is the *portio clavicularis* of the right trapezius. This latter muscle consists only of the middle portion, taking its origin from the seventh cervical to the fourth dorsal spine, inclusive, and being inserted into the acromion process and spine of the scapula. The lower third of the trapezius, below the fourth dorsal spine, is absent (see fig. 3).

The sensibility of the mouth, pharynx, and larynx is normal. The right vocal cord is immobile in the middle line; the left cord moves freely. The right omohyoid, stylohyoid, and posterior belly of digastric are absent. The right side of the tongue is smaller than the left (see fig. 2), and the tongue on protrusion points to the right side. (Part of this deviation is due to the obliquity of the floor of the mouth from deformity of the lower jaw.) The tongue can be moved freely in all directions. The right sternohyoid, sternothyroid and thyrohyoid muscles are absent. All of the above-mentioned muscles which are

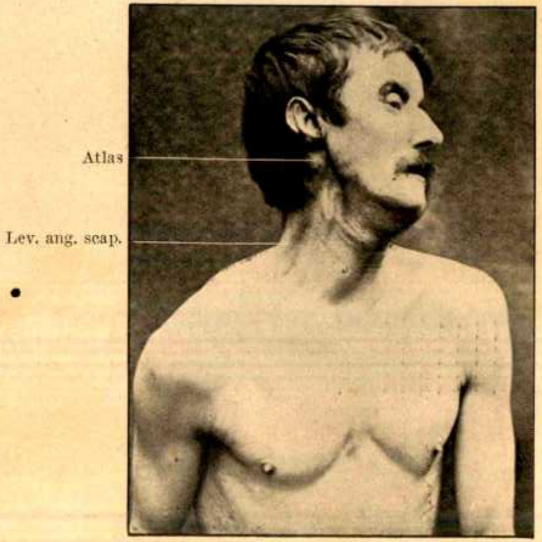

FIG. 1.

Showing mal-development of right pinna. The bifurcation of the carotid is subcutaneous. The right mastoid process is absent. The bony projection behind the angle of the jaw is the transverse process of the atlas.

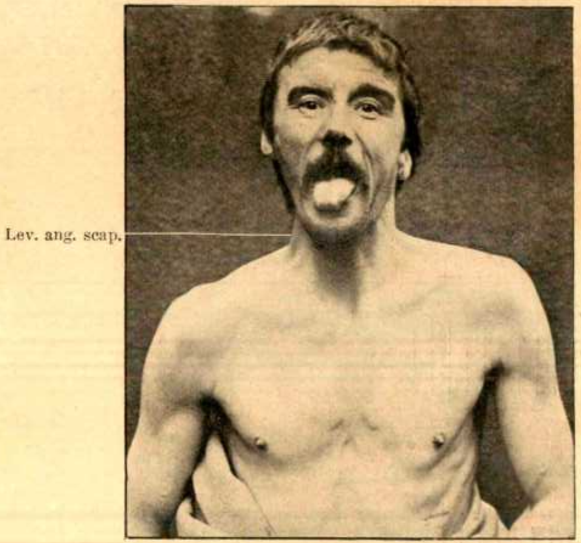

FIG. 2.

Showing deformity of right pinna and right side of lower jaw; also moderate atrophy of right side of tongue. Note absence of right sternomastoid and upper fibres of right trapezius. The right levator anguli scapulæ is subcutaneous.

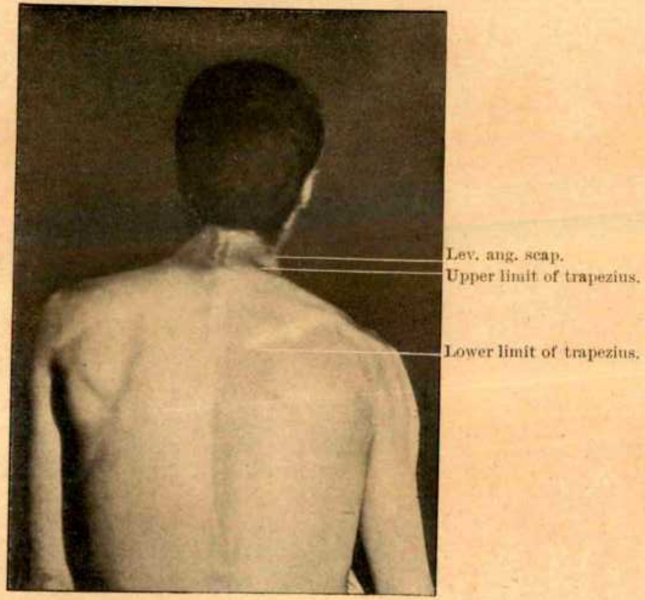

FIG. 3.

Showing downward and outward displacement of right scapula. Note parallel congenital white lines on back of neck. Right levator anguli scapulæ is subcutaneous. Right trapezius is absent above seventh cervical and below fourth dorsal spines.

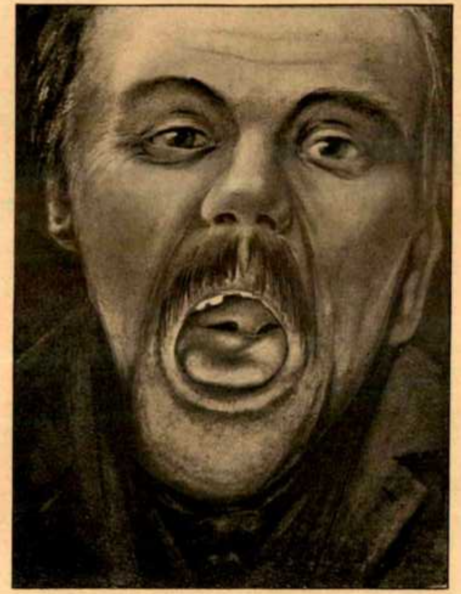

FIG. 4.

Photograph of soft palate during phonation. Note also deformity of lower jaw and of right pinna.

clinically non-active, are also non-responsive to the strongest electric stimulation, faradic or galvanic. Both sides of the tongue react normally.

In connection with the absence of the right sternomastoid, it is observed that the mastoid process on that side is absent and the transverse process of the atlas is subcutaneous. There is thus a deep hollow behind the angle of the right mandible, where the carotid vessels are visible, the bifurcation into internal and external carotids being easily seen (see fig. 1).

The levator anguli scapulæ is subcutaneous on the right side (see fig. 3), so also is the scalenus medius (see fig. 1). The right scapula is displaced downwards and outwards, its inner border running obliquely from below upwards and outwards, thereby rendering the outward displacement of its acromial angle still more marked. There is a very slight backward projection of the lower angle at rest.

On the back of the neck, from the level of the scalp line to that of the lowest cervical spine, there are two parallel rows of white, flattened, papillomatous projections, congenital, one row on each side of the middle line (see fig. 3).

The rhomboid muscles, the latissimus dorsi and all the other muscles of the right upper limb, together with the diaphragm, the muscles of the trunk and of the remaining limbs, are normal. The gait is normal. There is no anæsthesia. The supinator-jerks, knee-jerks, and ankle-jerks are normal, and the plantar reflexes are flexor in type. The sphincters are perfect.

In spite of the absence of so many muscles on the right side of the neck, all movements of the head and neck are executed powerfully on both sides, and there is no deviation of the head at rest.

CLINICAL DIAGNOSIS.

The absence of the right sternomastoid and of part of the trapezius, together with the paralysis of the palate and vocal cord on the same side, pointed to a congenital lesion of the spinal accessory and vagus nuclei on the right side. (This description of the spinal accessory nucleus is based on the modern nomenclature, maintaining that what was formerly termed the "bulbar part of the spinal accessory" is properly to be reckoned as part of the vagus nucleus.)

The absence of the mastoid process was probably the result of non-development of the sternomastoid muscle. The partial hemiatrophy of the tongue indicated an implication of the hypoglossal nucleus. The absence of the depressor muscles of the hyoid bone and of the posterior belly of the digastric indicated that the lesion extended down the anterior cornu of the spinal cord at least as far as the third cervical segment. This diagnosis of unilateral affection of the medulla and upper part of the cord was further supported by the maldevelopment of the right mandible and the absence of molar teeth in that bone, possibly attributable to an affection of the spinal root of the trigeminal nucleus. The other bulbar nuclei, from the facial upwards, were apparently unaffected.

The patient's unexpected death from pontine hæmorrhage, six weeks later, afforded an opportunity for pathological investigation of the case.

Subsequent history.—One morning the patient, who had not been complaining of headache or any other premonitory symptom, was sitting up in bed. Suddenly he fell back unconscious. When seen a few minutes later by the house physician, Mr. S. N. Crowther, he was found to be comatose, cyanosed, and breathing stertorously. The right arm and fingers were flaccid and motionless, whilst with the left he made semi-purposive clutching movements. The left lower limb was drawn up at the hip and knee. There was conjugate deviation of the eyes to the left. The knee-jerks were brisk and equal, there was ankle-clonus on both sides, and the plantar reflexes were extensor in type. The urine, drawn off by catheter, contained a large amount of albumen and a few granular casts. The temperature was 98° F. Two minims of croton oil were placed on the tongue.

When seen by me three hours later, the patient was still comatose and breathing stertorously, making occasional movements with the left hand when stimulated by pinching the limb. Occasionally the right elbow made movements of extension. There was no movement of the other limbs, which were flaccid and extended at all joints. The eyes were still deviated to the left. The pupils were unequal, R. > L. semi-contracted and reacting to light. There was slight weakness of the right face, the nasolabial fold being shallower than on the left side, and the right ocular fissure wider than the left. The breathing was laboured, all the extraordinary muscles at the root of the neck being in action. The diaphragm was hardly felt to contract at all, respiration being chiefly costal in type. The knee-jerks were brisk and equal, there was ankle-clonus, R. > L., the supinator-jerks were not elicited. The plantars were now absent, also the abdominal and epigastric reflexes. The pulse was hard and bounding, 132 per minute. There was some œdema over the lumbo-sacral vertebræ, none of the ankles. The temperature was 98·4° F. Lumbar puncture withdrew clear fluid, which contained no excess of leucocytes on microscopic examination. With the sodium hypobromide test a small amount of urea was detected in the fluid.

The patient died, without regaining consciousness, twenty-four hours after the onset of coma.

AUTOPSY.

The heart was hypertrophied and the kidneys granular, the pelvis of the right kidney being dilated. There was some recent hypostatic pneumonia, and at the right apex some old pleuritic thickening.

All the muscles of the body were well developed, except on the right side of the neck, where there was total absence of the *sternomastoid*, *sternohyoid*, *sternothyroid*, *thyrohyoid*, *stylohyoid*, *digastric* (posterior belly), and *omohyoid*. The only muscle superficial to the carotid vessels was the platysma, which was well developed. The upper fibres of the *trapezius* were absent, the middle fibres, arising from the seventh cervical to the fourth dorsal spine inclusive, were well developed, and below that level the muscle was absent. No signs of the spinal accessory nerve could be found. The rhomboids, levator anguli scapulæ, splenius and all other muscles of the neck were normal. The diaphragm was normal on both sides. The right *soft palate* was thinner than on the left side, and subsequent microscopic examination showed that on the paralysed side the *levator palati* was absent and the *azygos uvulæ* diminished in size. The right side of the tongue was smaller than the left. The epiglottis was bent over towards the right side and its right edge curved longitudinally.

The larynx was removed for more detailed examination. For most valuable aid in its dissection I desire to express my indebtedness to my colleague, Mr. James Black, lecturer on anatomy in the Westminster Hospital Medical School. The following are the chief points thus established:—

The thyroid cartilage, with its pomum Adami, was thrust across to the right side, giving the larynx a fictitious appearance of being more developed on the defective side. As a matter of fact the larynx was generally atrophic on the right side. This was noticeably the case with the right half of the cricoid cartilage. Subsequent dissection displayed the absence or most rudimentary condition of some of the muscles of the right side of the larynx.

Muscles.—The mylohyoid, geniohyoid and anterior belly of the digastric were about equally developed on the two sides. The posterior belly and the intermediate tendon of the last-named muscle were attenuated on the right side, and represented only by a faint strand of fibrous tissue. The right hypoglossus was well developed, but somewhat smaller than on the normal side. The right middle constrictor of the pharynx was very defective, and the styloid muscles could scarcely, if at all, be recognised. The intrinsic muscles of the tongue appeared generally fairly symmetrical on the two sides. *Muscles of the larynx.*—The right cricothyroid was totally absent. This, together with the absence of all the right infrahyoid muscles as already described, rendered the origin of the inferior constrictor of the pharynx strikingly apparent. The right crico-arytenoideus posticus was absent, and the right crico-arytenoideus lateralis represented by a thin film of pale muscle fibres, too rudimentary to be capable of function. The small muscle described by Merkel as the kerato-cricoid, however, was present. Viewing the posterior surface of the cricoid cartilage, its asymmetry was very marked, the well-developed left crico-arytenoideus posticus presenting a noticeable contrast to the bare area of cartilage on the defective side. The arytenoideus transversus and obliquus were small but otherwise presented no feature for criticism. The right thyro-arytenoideus was but feebly developed, and this was well brought out on examining a coronal section of the larynx, when the meagre bulk of the prismatic sectional area of the right vocal cord contrasted strikingly with that of the opposite side.

Nerves.—A most careful search failed to discover any trace of the right hypoglossal nerve. The right lingual nerve was present, and a number of branches passed from it into the hypoglossus muscle (corresponding to those which usually form loops of communication with the hypoglossal nerve on this muscle). The right superior laryngeal (internal laryngeal branch), and the right recurrent laryngeal nerve, although smaller than on the left side, were recognised.

The foramen cæcum on the dorsum of the tongue was much deeper than usual. There was a well-marked lingual lobe to the thyroid gland, with a long pedicle leading upwards towards the root of the tongue, but no actual connection could be demonstrated between this pedicle and the lower end of the foramen cæcum.

The right external auditory meatus was shorter and narrower than the left, but the tympanum and auditory ossicles were normal.

The cerebrum showed no signs of disease on naked-eye examination, apart from an agonal thrombosis of the left middle cerebral artery, unassociated with any area of softening. The left cerebral hemisphere was larger than the right, weighing 18 ozs. as against 15 ozs. The left cerebellar hemisphere was also slightly wider from side to

The pons on naked-eye examination was symmetrical. On cutting into it, after hardening in Müller's fluid, several small hæmorrhages were found, of which the largest was about the size of a pea, in the upper third of the pons, dorsal to the transverse fibres, in the region of the fillet and posterior longitudinal fasciculus and in the intermediate area between these structures (see figs. 5 and 6). The remainder of the pons was normal and symmetrical on microscopic examination.

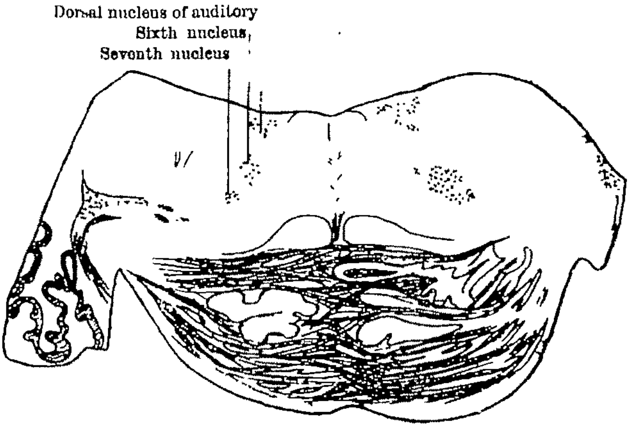

Fig. 7.

Pons.—Level of emergence of auditory nerve.

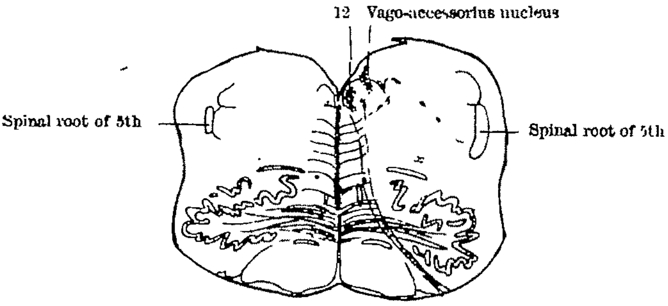

Fig. 8.

Medulla.—Upper.

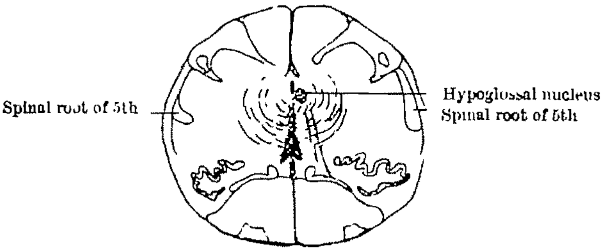

FIG. 9.

Medulla.—Lower.

The medulla oblongata showed marked abnormality, as seen in figs. 8 and 9. The olivary bodies were normal and symmetrical. On the right side the hypoglossal nucleus and the adjacent lower part of the accessorio-vagus nucleus were absent, whilst the corresponding nuclei on the left side were well developed. A few cells were present in the position of the uppermost part of the right hypoglossal nucleus, but they were of a smaller type than those of the left hypoglossal nucleus, and apparently represented part of the vagus nucleus, displaced towards the middle line. The spinal root of the trigeminus, capping the substantia gelatinosa, was much smaller on the right side than on the left. This latter abnormality did not extend down into the cord. The other nuclei of the medulla were normal, likewise the various tracts, ascending and descending.

In the spinal cord the anterior median fissure was expanded into a deep cleft, extending from the lower part of the second cervical to the upper part of the seventh cervical segment. Along the right margin of this wide fissure ran the anterior spinal artery. The cleft was lined by a thin layer of pia mater. It penetrated deeply into the gray matter on the right side, near the junction of the anterior cornu with the gray commissure (see fig. 10). The gray matter of the cord was thus much distorted, and the asymmetry could be traced upwards above the level of the actual cleft to the first cervical segment and on into the medulla, as already described. Below the seventh cervical segment the cord showed no abnormality. The nerve-roots, anterior and posterior, were given off from the gray matter on both sides, including the cervical region. A

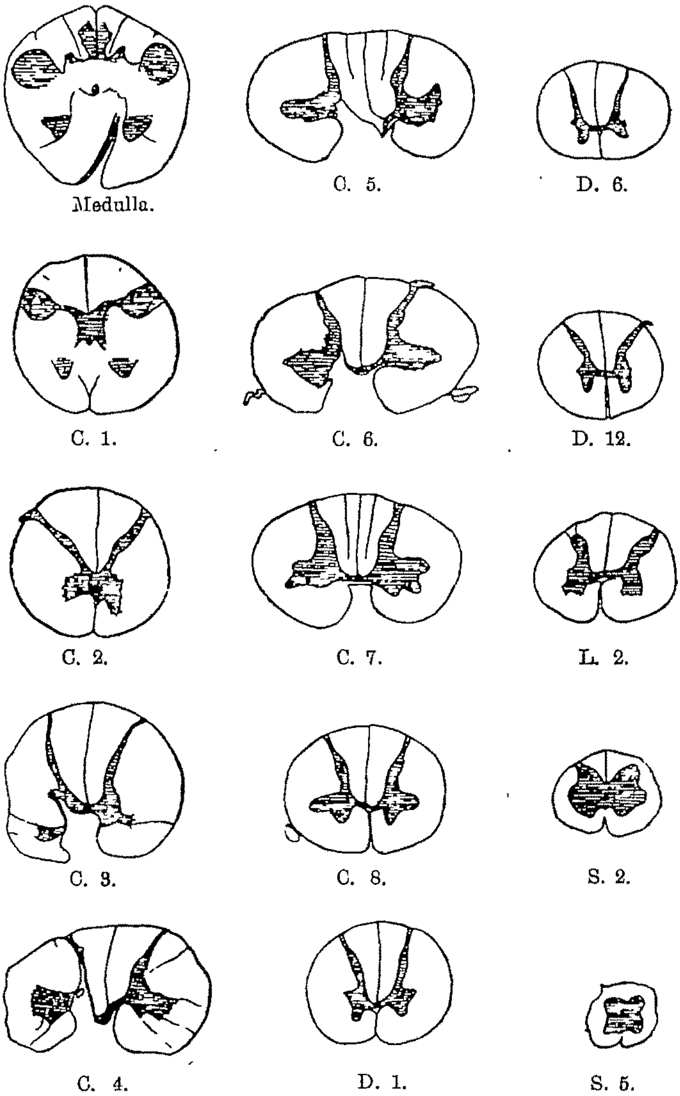

Fig. 10.

Medulla and spinal cord.

small number of nerve-cells in the right anterior cornu were missing in the position of the cleft. The distortion of the remainder of the gray matter, however, rendered it impossible to identify precisely in each segment the particular groups of cells which were absent.

The points of interest in this case, clinically and pathologically, are numerous.

Firstly, as to the fatal hæmorrhage. This, a secondary result of chronic renal disease, was situated in the tegmental region of the upper part of the pons, located almost mesially, but more extensive on the right side of the middle line (see figs. 5 and 6). It was extra-pyramidal. Clinically, the signs were somewhat confusing. The lower limbs were flaccid, yet there was well-marked ankle-clonus on both sides, together with an extensor type of plantar reflex. There were semi-purposive irregular movements of the right upper limb, and slight facial weakness on the right side, together with conjugate deviation of the eyes to the left. These two latter signs would have been explainable by a lesion implicating the right sixth nucleus. But the autopsy showed the nucleus to be intact. The ocular deviation, therefore, is more probably attributable to an interruption of the fibres connecting the right sixth with the left third nucleus, as in a somewhat similar case recorded by Raymond and Cestan (7).

The condition of the reflexes, and especially the presence of the extensor type of plantar reflex, is of especial interest, for in this case there was no interruption to the pyramidal tracts in any part of their course, the hæmorrhage being situated posteriorly in the tegmentum. Kornilow (5) has recently maintained that the Babinski phenomenon can occur independently of a lesion of the pyramidal tracts, and this case supports such a view. The explanation of its occurrence, however, remains obscure. Another unusual feature was the absence of the high temperature which so frequently occurs in fatal pontine hæmorrhages. Nor were the pupils contracted.

Even more remarkable is the unilateral lesion in the medulla oblongata and upper part of the spinal cord. The condition was congenital and non-progressive. It does not correspond to any variety of syringomyelia. I have been unable to find in the literature of nuclear lesions of the medulla or cord the record of anything precisely similar. The two parallel white lines visible in the skin of the back of the neck (see fig. 3), were probably associated developmentally with the lesion of the brain-stem. They may be regarded as analogous to the mole in the lumbar region, so commonly seen in cases of spina bifida occulta.

Only a small number of cases (chiefly of syringomyelia) have been published, *e.g.*, those of Schmidt (10), Kretz (6) and Weintraud (14), in which the diagnosis of nuclear paralysis of the accessorius was made, and in none of them was the case completed by subsequent pathological investigation.

In our case the lesion of the central nervous system was an extensive one, implicating the gray matter of the following motor nuclei :—vagus, accessorius, hypoglossal, and anterior cornu as low as the sixth cervical segment. Together with these, the spinal root of the trigeminus was also affected. The other cranial-nerve nuclei, from the facial upwards, were unaffected. On the affected side the spinal accessory and hypoglossal nerves were absent, whilst the anterior cornu, though distorted in shape and diminished in size, gave off its anterior nerve-roots throughout the affected region. The vagus trunk was present, though smaller on the affected side, its superior laryngeal and recurrent laryngeal branches being especially reduced in size.

This case is also of value in regard to the innervation of of the trapezius and as to the parts played by the spinal accessory and by the cervical nerves respectively. In our patient the spinal accessory nerve was absent, the cervical nerves present. Associated with this there was total absence of the sternomastoid and of the upper and lower thirds of the trapezius, whereas the middle third of the trapezius was well developed. This indicates a separate innervation of the middle third of the trapezius by the cervical anterior roots, the remainder of the muscle belonging to the territory of the spinal accessory. Such a view harmonises with the opinion of Remak (8). Gowers (4), in his latest edition, states that the whole of the trapezius is supplied by the spinal accessory, and that the twigs from the cervical plexus merely pierce the muscle without supplying it. Schulz (11), again, from a study of a large number of recorded cases, comes to the conclusion that the result of a pure lesion of the spinal accessory, without implication of the cervical plexus, is complete palsy of the lowest third and partial palsy of the upper two-thirds, the clavicular fibres being more severely paralysed than the acromial. The apparent divergence of these views is probably to be explained by variations in the proportional territory of the spinal accessory in different cases. Thus, for example, Schlodtmann (9) records an interesting case of unilateral paralysis of the palate, larynx, sternomastoid and trapezius, the result of a fracture of the base of the skull, without implication of the cervical roots, and in which there survived only a few strands of the acromial bundle, whose total breadth was about that of a pencil.

The characteristic deformity occurring in most cases of trapezius paralysis was clearly described by Duchenne (3), who applied to the displacement of the scapula the term "*mouvement de bascule,*" and stated that the occasional absence of this deformity in certain cases was explainable by survival of the acromial bundle, quite a small strand of this bundle being sufficient to prevent the displacement. But in our case the acromial bundle was well developed, and yet the scapular displacement was perfectly evident.

As to the sternomastoid, it should be noted that in our case its absence produced neither deformity at rest nor impairment of the movements of the head and neck. This exemplifies the truth of Gowers' dictum (4) that there is no such thing as a "paralytic" torticollis, though Bernhardt (2) in his recent text-book states that unilateral paralysis of this muscle causes moderate deviation of the head towards the affected side, the chin being slightly raised. In our case no such deviation occurred.

Similarly, the absence of all the infrahyoid muscles produced no impairment of movement of the hyoid bone, though the larynx was distorted towards the affected side.

It is interesting to note that, in spite of the absence of the right hypoglossal nucleus and nerve, the corresponding hemiatrophy of the tongue was only moderate in degree. The intrinsic muscles of the tongue were certainly smaller on the right side than on the left, but the tongue itself was not curved longitudinally as in ordinary hypoglossal palsy, nor were its electrical reactions different on the two sides. Moreover the orbicularis oris was unaffected, contrary to what is usually found in bilateral lesions of the hypoglossal nuclei.

The explanation of these facts is somewhat difficult. Most likely the hypoglossal nucleus of the healthy side controlled both halves of the tongue. (The right and left nerves, according to Thane (13), are occasionally connected by a cross branch or loop in the substance of the genio-hyoid.) Another less probable view might be that, inasmuch as the lingual nerve supplied branches which entered the hyoglossus, the motor fibres of the trigeminus may have taken on a vicarious distribution.

Nor is the question appreciably enlightened by a consideration of the absence of the depressor muscles of the hyoid bone. Whichever nerve assumed the vicarious function of innervating the right half of the tongue, did not reach the infrahyoid muscles. These latter are commonly believed to be supplied, not by the hypoglossal itself, but by branches from the first and second nerves forming the loop of the *ansa hypoglossi*. Thus Beevor and Horsley (1), who stimulated the intracranial roots of the hypoglossal in the monkey, obtained no contraction in the infrahyoid muscles, which responded briskly, however, to stimulation of the first and second cervical nerves. Yet Stierling (12), on the other hand, records a case of annular fracture of the anterior part of the foramen magnum in man, implicating the hypoglossal nerve close to its origin, in which, together with paralysis of the vagus on that side, there was paralysis of the corresponding half of the tongue and of the depressors of the hyoid bone on the same side, the cervical roots being stated to be uninjured.

In our case there was a deep fissure in the anterior cornu, which was most probably causally connected with the absence of the infrahyoid muscles. And yet all the anterior roots were present in the cervical region, and, moreover, were efficient in supplying the other deep muscles of the neck, together with the middle third of the trapezius. Of course, it must be remembered that the nucleus of the spinal part of the trapezius lies lower in the cord than that of the infrahyoid muscles.

The atrophy of muscles in the larynx is undoubtedly to be associated with the lesion of the vagus, the lower part of whose nucleus was deficient, and whose trunk was much diminished in size, especially its superior laryngeal and recurrent laryngeal branches. Of the laryngeal muscles certain were absent (crico-thyroideus and crico-arytenoideus posticus); others were so rudimentary as to be incapable of function (crico-arytenoideus lateralis); others, again, were simply diminished in size (arytenoideus and thyro-arytenoideus). The position of the vocal cord corresponded to that usually seen in total recurrent laryngeal palsy, but there could be no question of contracture of abductor or adductor muscles, since the crico-arytenoideus posticus was absent and the crico-arytenoideus lateralis reduced to a rudimentary strand. The position of the cord was simply that of the edge of the thyro-arytenoideus muscle, which supported the cord immovably in a semi-abducted posture.

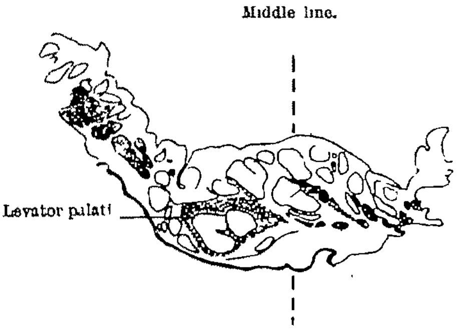

FIG. 11.

Soft Palate.—Showing absence of levator palati on right side.

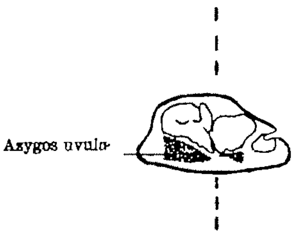

FIG. 12.

Uvula.—Showing atrophy of azygos uvulæ on right side.

In the soft palate the right levator palati was absent, and the right azygos uvulæ was much smaller than the left (see figs. 11 and 12), whilst in the pharynx the right middle constrictor was greatly diminished in size. This corresponds with remarkable accuracy to a lesion of that part of the vagus formerly named the "bulbar part of the spinal accessory." Beevor and Horsley (1) have shown experimentally in the monkey that the motor fibres for the levator palati and azygos uvulæ, as well as part of the constrictor muscles of the pharynx, are thus supplied.

The deformity of the temporal bone and lower jaw is also deserving of notice. Absence of the sternomastoid is sufficient to account for the rudimentary condition of the mastoid process. The smallness of the lower jaw, however, cannot be explained as the result of muscular atrophy, since all the muscles attached to the mandible were present and well developed. Probably the condition of the lower jaw is to be associated with the congenital smallness of the spinal root of the trigeminus. Such a view is corroborated by the absence of the lower molar teeth and by the smallness of the external auditory meatus and pinna on the affected side. The external auditory meatus, pinna, middle ear and Eustachian tube are all developed from the first visceral cleft, whilst from the mandibular arch, between this cleft and the mouth, the lower jaw is developed. And all these structures lie within the territory of the trigeminal nerve.

To Dr. Ferrier I desire to express my indebtedness for his permission to conduct the microscopical portion of the above research in the neuro-pathological laboratory of King's College.

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BINOCULAR AND STEREOSCOPIC VISION IN MAN AND OTHER VERTEBRATES, WITH ITS RELATION TO THE DECUSSATION OF THE OPTIC NERVES, THE OCULAR MOVEMENTS, AND THE PUPIL LIGHT REFLEX.

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THE work on which this paper is based has been done at intervals during the last seven years, and consists partly of the results of a series of experimental lesions of the optic tract on one side in cats; of division of the posterior commissure in cats; and the division of one optic nerve in pigeons, owls, rabbits, and cats, followed by microscopical examination by the Marchi method of the optic chiasma and optic lobes in these animals, and by the Weigert-Pal method in the toad and chameleon. In addition, I have studied the pupil reaction to light, the position of the eyes in the head, and the ocular movements in a considerable number of animals, and the conclusions at which I have arrived I have drawn up in the following summary, as a preface to the paper.

SUMMARY OF CONCLUSIONS.

(1) The decussation of the optic nerves at the chiasma is complete in all fishes, amphibia, reptiles and birds, whether possessed of binocular vision or not.

(2) Binocular vision is originally associated with carnivorous habits, and is found to a moderate degree amongst carnivorous fishes in a few of the sharks and rays, in some amphibia, as the toad, which lives on flies and insects, and

in many carnivorous birds, especially the larger gulls, some penguins, hawks, owls and vultures. Amongst mammals binocular vision is especially developed in the carnivora and in the primates. In the latter group of monkeys and man perfect binocular vision is probably associated with the development of the hand as a prehensile organ.

(3) Though many of these animals have fair binocular vision, yet in all vertebrates below mammals there is total decussation of the optic nerves at the chiasma, and their vision is therefore not stereoscopic in the same sense as in the higher mammals, in which visual impressions from the two eyes are received on the same side of the brain, owing to the semi-decussation of the optic nerves at the chiasma; and their macular vision is also less developed than in the higher mammals, as in the felidæ and primates.

(4) In animals with laterally-placed eyes and periscopic vision the movements of the eyes are independent of each other, as seen typically in the chameleon, while conjugate movements of the eyes for lateral and vertical movements are associated with the acquirement of stereoscopic vision. Convergence of the eyes in the act of feeding is seen in many animals with total decussation of the optic nerves and otherwise independent movements of the eyes, as in the chameleon and the hornbill.

(5) The reflex contraction of the pupil to light is limited to the eye stimulated, and is not consensual, in amphibia and in birds, whether possessed of binocular vision or not. In the rabbit, with scarcely any binocular vision, only the one pupil reacts, and the decussation is almost complete. In cats and in the higher mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there is consensual pupil reaction. Consensual pupil contraction to light is therefore dependent, not on binocular vision alone, but on semi-decussation of the nerves at the chiasma.

(6) In the rabbit and the cat both the crossed and the uncrossed fibres are spread equally through all parts of the two tracts, and are not collected into any distinct bundle.

(7) Experimental division of the optic tract on one side

in cats produced distinct homonymous hemianopia, with the hemianopic pupil reaction, and with considerable diminution of the pupil reaction to direct light in the opposite eye, though its consensual reaction was brisk on exposure of the other eye to light.

(8) Injuries, either lateral or dorsal, of the anterior part of the anterior corpus quadrigeminum in cats, if deep enough to wound the tegmental roof surrounding the central gray matter, have produced in four cases descending degeneration in Meynert's fountain decussation, most of the fibres decussating at the level of the third nerves to form a longitudinal tract immediately ventral to the opposite posterior longitudinal bundle, but a few uncrossed fibres passing into the posterior longitudinal bundle of the same side. These are the antero-lateral columnar fibres already traced by Boyce into the lower cervical cord. In no case have I produced any degeneration in Forel's lower decussation.

(9) In animals with divergent optic axes and periscopic vision and total decussation of the optic nerves there must exist a posterior total decussation of the pupil reflex fibres connecting the anterior corpora quadrigemina with the third nuclei; while in mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there must be a semi-decussation of the posterior pupil reflex fibres. Consensual reflex contraction of the pupils to light is effected by means of this posterior semi-decussation between the corpora quadrigemina and the third nuclei, and not by means of any commissural fibres between the sphincter pupillæ centres in the third nuclei.

(10) The lesion in typical Argyll-Robertson pupil is probably a sclerosis of these fibres, some of the fibres of Meynert's decussation, and simple reflex iridoplegia is not due to a nuclear lesion, though a nuclear lesion may be superadded in the dilated type of Argyll-Robertson pupil, in which the reaction to convergence and accommodation is also lost.

I have divided my paper under six headings:—

(1) Binocular vision, and its relation to the decussation of the optic nerves, and to the feeding habits of the animal.

(2) Stereoscopic vision.

(3) Macular vision.

(4) Ocular movements, conjugate and independent, and their relation to stereoscopic and periscopic vision respectively.

(5) The path of the pupil reflex to light, including the results of experimental section of one optic nerve, one optic tract, and of the posterior commissure in cats.

(6) Argyll-Robertson pupil.

The experimental part of this work was commenced in 1897, in order, by studying the path of the pupil reflex to light in various animals, to afford some help in elucidating the problem of reflex iridoplegia, or the Argyll-Robertson pupil. Incidentally, some other interesting facts have been observed, bearing on the relation of binocular vision in birds and animals to their feeding habits and mode of life. Some of the contents of this paper were read at a special meeting of the Neurological Society on December 21, 1899, when the microscopical sections relating to the experimental part of the work were shown. Publication has been delayed until now in the hope of rendering the work more complete in a larger series of animals; especially I wished to obtain a specimen of the optic chiasma of the fox shark, *Alopecias vulpes*, sometimes known as the thresher, which is remarkable amongst elasmobranchs for possessing a fair degree of binocular vision, the eyes being set far forwards on the head. I have not, however, been able to obtain a specimen. An ideal experiment would be an experimental enucleation of one eye in such an animal, with subsequent tracing of the degenerated nerve-fibres through the optic chiasma and tracts, by Marchi's method, as Krause (14) did with goldfish. The difficulties of attaining this end, however, appear to be even more insuperable.

BINOCULAR VISION.

Knowing the rashness of assuming anything to be true without proof, yet, in the absence of direct evidence, I think it may be safely assumed that the decussation of the optic

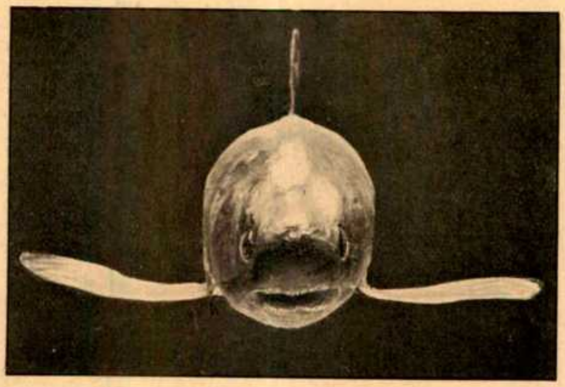

FIG. 1.

Front view of fox shark, *Alopecias vulpes.*

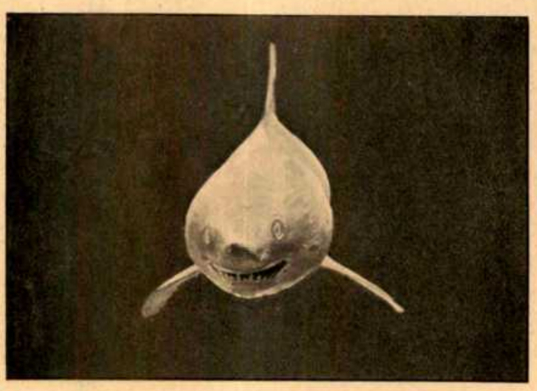

FIG. 2.

Front view of Porbeagle shark, *Lamna cornubica.*

animal. Stuffed specimens are apt to be deceptive in regard to the true position of the eyes of the animal during life, a greater or less amount of stuffing of the head altering the angle of apparent vision considerably. Making all due allowance for this, however, it seems clear that partial binocular vision must be admitted for these species of elasmobranch fishes.

The presence of binocular vision in animals seems to me to depend upon their feeding habits rather than upon their social position, if I may use the term, in the animal scale. Carnivorous animals, which have to follow up and catch their prey, which is likely to be dodging hither and thither in the attempt to elude their captor, require especially keen sight for this purpose, and it is manifestly an advantage for them to be able to keep both eyes fixed at the same time upon the moving object. To effect this, their eyes are set well forwards, allowing of more or less binocular vision. Amongst fishes this arrangement is found in only a few of the elasmobranchs, carnivorous fishes with rapid power of locomotion. It is not found by any means in all of these carnivorous fishes, even among the fiercest and most active, such as the blue shark, *Charcarodon*, and in the more slowly moving skates and rays the eyes are set completely back to back. It seems not improbable that in this primitive group of vertebrates the extra value of binocular vision to a carnivorous animal, which has to pursue and catch its prey, is only commencing to be recognised, a few types only having acquired it, and that to an imperfect degree. Amongst the amphibia a higher degree of binocular vision is seen in the frogs and toads, the total divergence of the optic axes varying from about 135° in *Rana clavata* to 120° in the bull frog, while in the giant toad, *Bufo marinus*, the angle is as small as 90°.

In the chameleon is seen an extraordinary combination of completely independent movements of the two eyes for all ordinary purposes of vision, such as when seeking for its prey (flies, grubs, worms, &c.), with the most perfect power of convergence of the eyes, with binocular vision, in the act of striking its prey. This animal has the most extended range of movement of the eyes of any animal I have studied. Each eyeball is almost completely enclosed in a casing of the ordinary horny epidermis which covers the whole animal, leaving only a small round aperture in front, through which can be seen, deeply placed, the small pupil. The visual field of each eye must be therefore very small and constricted, but to make up for this the range of movement of the eyes is very great, the eyeball being capable of being turned through about 180° in the horizontal plane from directly forwards to a backward direction, and through about 90° in the vertical plane. The eyes move in a slow, jerky fashion, recalling the movement of a searchlight, while the visual field of each eye must resemble the limited field illuminated by a searchlight, on account of the deep setting of the eye. Except when the animal is in the act of striking its prey, when fairly active, the eyes are continually being moved about, but quite independently, one eye perhaps looking forward, and the other pointed downwards and backwards. Under these circumstances I have never seen either eye point further forward than up to 15° of the antero-posterior line, and thus I first came to the erroneous conclusion that this animal had no power of binocular vision, since the animal I first studied would not feed, it being then late in the autumn. I have, however, since studied the movements of the eyes in a feeding animal, and it was a great surprise to me to see how perfect a power of convergence of the eyes the chameleon possesses. If, while the animal is at rest, and the eyes are roving in different directions independently as usual, a mealworm is offered within striking distance, that is to say, about the animal's own length away, presently one eye will catch sight of it, and if the bait is acceptable the head will be slowly turned towards it, and both eyes will be brought to bear upon the morsel, actually converging directly upon it. The mouth is seen then slowly to open slightly, and the moist tongue shows between the animal's jaws, when, like a flash, the tongue is thrown out and seizes the worm, and is retracted again into the mouth. The first part of the operation is performed slowly, with the deliberation charac-

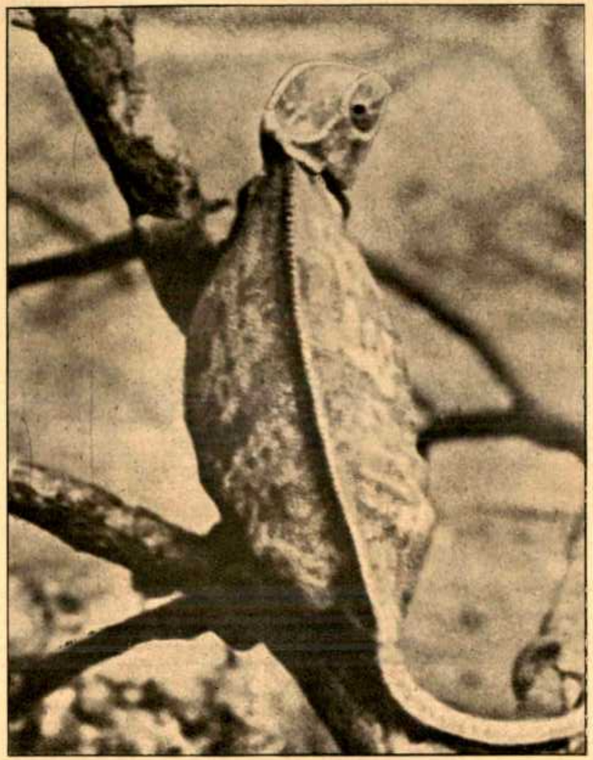

Fig. 3.

Chameleon, back view, showing the right eye turned directly backwards towards the camera.

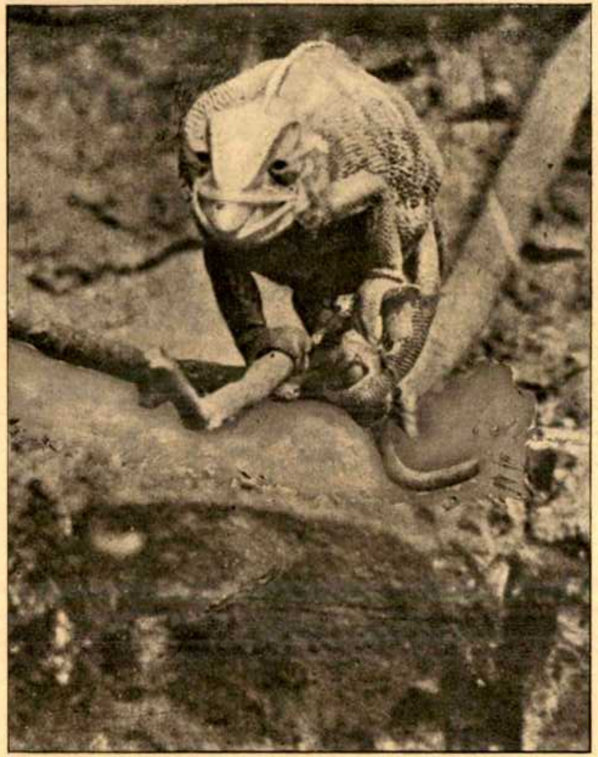

Fig. 4.

Chameleon, front view, showing the eyes converged on a mealworm (not shown in photo), while in the act of striking with the tongue.

tunity is not likely to be afforded him under natural conditions, and it is obvious that the focussing of both the eyes upon the object, giving him binocular vision, is of great advantage to him in the accomplishment of this accuracy of range finding.

Although this degree of binocular vision is found amongst certain of the amphibia and reptiles, yet the optic nerves in these animals decussate totally at the chiasma, as I have shown by cutting sections of the chiasma in a toad and in chameleon (fig. 5). In birds, much greater variations are met with in the positions of the eyes in the head than is seen in reptiles, amphibia and fishes. In graminivorous and fruit-eating birds, as the parrots, pigeons, fowls, ducks, swans, many finches, and others, the eyes are set laterally on the head, no attempt at binocular vision being possible. Indeed, when such a bird attempts to look at any object carefully, it always holds its head, as it were, on one side. Birds more omnivorous in their feeding, such as the robin, thrush, and sparrow, have slight binocular vision, the eyes being set more forward. It is in the birds of more purely carnivorous habits, which require especially keen sight to follow up their prey, that the highest degree of binocular vision is seen, with the eyes set further forward, and more side by side. Amongst fish-eating birds this is

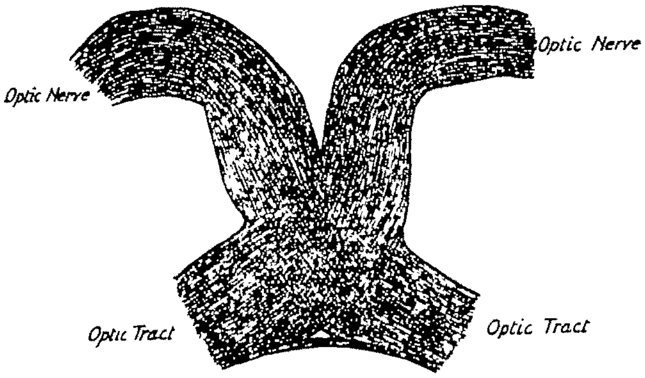

FIG. 5.
Section of chiasma of chameleon, stained by Weigert-Pal method.

well seen in the heron, kingfisher, bittern, puffin, penguins, and the larger gulls. Indeed, the gannet, of the fish-eating gulls, and the thick-billed penguin have as much binocular vision as some owls. Considerable binocular vision is also seen in the shrike, or butcher-bird, and in the goat-sucker, or night-jar. This latter bird has a head very like a small owl, and it also resembles the owl in its feeding habits, preying on moths and cockchafers in the dusk of twilight. The class of birds which shows the most consistent amount of binocular vision is the raptatores, or the hawks and harriers, vultures and owls, and of these the owls have by far the greatest degree of binocular vision; in some of them, as in the great eagle owl, *Bubo maximus*, the total divergence of the optic axes is no greater than 45°. The reason for this great development of binocular vision in the owl is apparent when we compare his feeding habits with that of other raptatores. Watching in the dusk for field-mice and other small animals on which he preys, the accuracy of sight and range due to his great binocular vision must be of immense advantage in enabling him to follow the quick, jerky movements of such small mammals, and to pounce upon his prey with unerring accuracy in the dim light of twilight. Although the vision of a pigeon and of an owl is so different in character, the one having laterally placed eyes, an extended range of vision on both sides, and no binocular vision, while the latter has the eyes placed right forward, with accurate binocular vision, but with a less extended range, yet the decussation of the optic nerves is complete in both cases. In the pigeon I have followed the path of the degenerated optic fibres through the chiasma after enucleating one eye, and a fortnight later killing the bird, and cutting sections of the chiasma after staining with Marchi's method, which showed that all the degenerated fibres crossed over into the optic tract on the other side (fig. 6). I attempted the same experiment in the owl, but failed three times, the bird in each case not surviving the operation long enough for the characteristic degeneration to take place. I therefore stained sections of the chiasma by Pal's method, which show quite clearly that the decussation of the fibres must be complete; the angle of entrance of the optic fibres being too sharp to suggest the possibility of any of them turning back into the tract of the same side, and all the visible bundles, though interlacing with those of the other optic nerve, appear to pass across into the opposite optic tract.

Turning to mammals, we find here again a similar variation in the position of the eyes in the head, laterally placed eyes being found for the most part in the herbivora and rodents, that is to say, in animals whose food does not run away from them, whilst the most perfect binocular vision is found in the carnivora, especially in the cats, and in the primates. Taking the rabbit or hare as a type of a rodent, its eyes are set quite laterally, with practically no binocular vision possible, giving the animal a wide range of vision on either side, which, as Lindsay Johnson suggests (12), must be of great service in giving early warning of the near approach of possible enemies. Yet this great range of lateral vision makes of necessity vision in the direct line ahead correspondingly bad, and it is well known how a rabbit or hare may run straight towards a gun, through inability to perceive the danger that lies directly in front of him. Associated with this lateral or periscopic vision is a

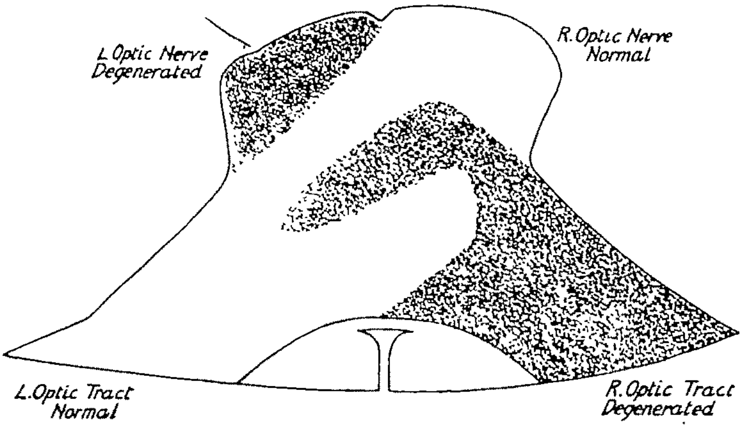

FIG. 6.

Chiasma of pigeon, stained by Marchi's metheod, after enucleation of the left eye.

practically complete decussation of the optic nerves at the chiasma. This I have examined both in the ordinary way by sections stained with hæmatoxylin by Pal's method, and also after experimentally enucleating one eye and a fortnight later staining with Marchi's method to trace the degenerated fibres. The Pal sections appeared to show total decussation, none of the fibres being seen to turn into the optic tract on the same side, though the angle formed by the optic nerves and the tracts at the chiasma is very much wider than in the owl, so that it is easy to conceive that in the rabbit a few fibres may turn into the optic tract on the same side. This indeed the Marchi sections show (fig. 7), as has also been shown by Ramón y Cajal (4), whose figures of the rabbit's and cat's chiasma stained by Marchi's method are exactly like the sections I exhibited at the Neurological

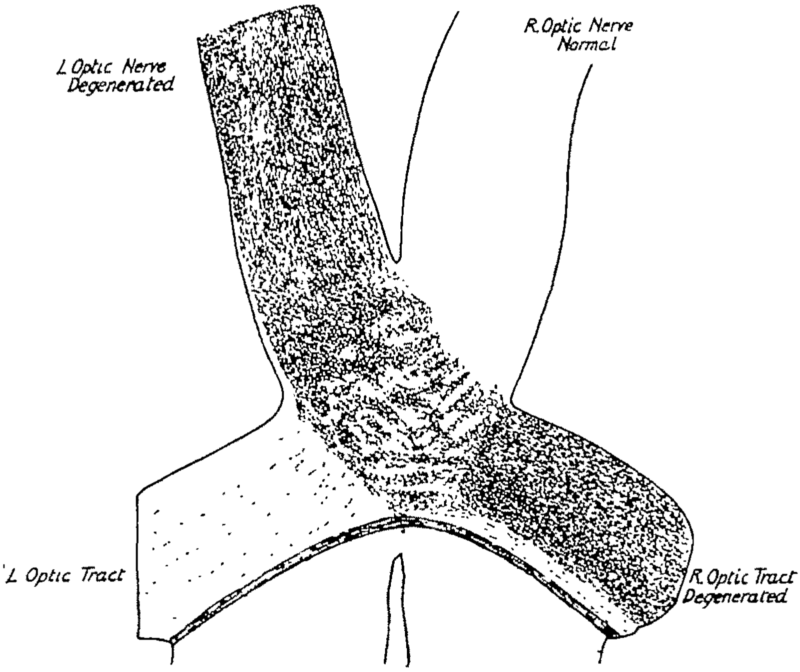

FIG. 7.

Section of chiasma of rabbit, stained by Marchi's method.

Society in 1899. According to Singer and Munzer (16) the decussation is complete in mice and rats.

Amongst the ungulata a certain amount of binocular vision is the rule, as in horses, oxen, antelopes and deer. The divergence of their optic axes is, however, great, and their acuity of vision is probably not very good, their senses of smell and hearing being proportionately keener. In these animals, although the binocular vision is not so well marked as in most of the owls, yet the movements of the eyes are mainly conjugate, not independent as in birds, and the decussation of the optic nerves is not complete, a small bundle turning into the optic tract of the same side. This has been shown in the horse and calf by two Vienna observers, using Marchi's method. Practically perfect binocular vision is seen amongst the feline group of the carnivora, cats, lions, leopards and tigers, predatory animals whose method, like that of the owl, is to lie in wait and pounce on their prey in the dim light. Other members of the carnivora, such as the dogs and foxes, bears and otters, on the one hand, and the seals, walrus and sea-lions on the other, have rather less marked binocular vision than the cats, the face protruding more between the eyes; this is, perhaps, most marked in some of the frugivorous bears. Anyone who has observed the habits of cats and dogs will, I think, admit that the cat has far the keener vision of the two, the dog depending more on scent. An additional point which confirms me in this opinion is the sluggishness of the pupil reaction to light in the dog as compared with the brisk contraction of the cat's pupil on exposure to light. This sluggishness of the pupil reaction to light is also seen in herbivora and rodents, such as horse, ox, rabbit, and it is almost certain that in these animals keenness of sight is less well developed than their acuity of hearing and smell perception.

The highest development of binocular vision is found in the anthropoidea, man, apes, and monkeys. In the lemurs it is less well developed, the face protruding more between the eyes, like that of a dog. It is only in the group of anthropoidea that high development of binocular vision is found unassociated with specially carnivorous habits, and its acquirement in such high degree is probably associated with the development of the hand as a prehensile organ, objects seized being submitted to the test of inspection rather than that of smell, a special sense that is becoming atrophic in the primates. Binocular vision is clearly of great assistance in the accurate use of the hand for fine movements, which, in proportion to its specialisation as a fine and delicate instrument, has been accompanied by increasing development of brain power.

STEREOSCOPIC VISION.

Although binocular vision is met with in many birds and a few fishes, and some amphibia and reptiles, yet none of these possess true stereoscopic vision, as is met with in the higher mammals, in which visual impressions from the two eyes are received on the same side of the brain, owing to the semi-decussation of the optic nerves at the chiasma. In a few mammals, as mice and other rodents, and in all the lower vertebrates, birds, reptiles, amphibia, and fishes, the decussation of the optic nerves is total, each optic nerve ending in the optic lobe of the other side. Visual impressions are further conducted to the cerebral hemisphere on the same side as the optic lobe which receives the impressions, for it has been shown in pigeons that such experimental extirpation of one cerebral hemisphere has been followed by complete blindness of the opposite eye; that is to say, psychical blindness, in which there is no definite conscious perception, though it is true that when both hemispheres have been removed it is clear that the pigeon is guided to a certain extent by reflex retinal impressions, not recognising food or the presence of an enemy, except so far as they are objects which reflect light, and so stimulate the retina. Edinger (6) states that in some birds, as the owl, the decussation of the optic nerves is only partial, but with this statement I cannot agree. Whether the opposite eye would be equally blind in an owl, which has fair binocular vision, as it has been shown to be in a pigeon, which has laterally placed eyes, after extirpation of the cerebral hemisphere on one side, has not to my knowledge been shown. The probability is that it would be the same as in a pigeon, since the decussation of the optic nerves seems to be as complete, and there is no known crossed afferent tract connecting the optic lobe of one side with the opposite cerebral cortex. It is therefore probable that in the owl and other birds with partial binocular vision the association of the visual impressions received by the two eyes of the same object is effected by the commissural fibres of the corpus callosum. Now, in the primates, and in less degree in other mammals with well-developed binocular vision, such as the felidæ, there is true stereoscopic vision, owing to the visual axes being approximately parallel, so that the two maculæ are brought to bear on the same object, and owing to the semi-decussation of the optic nerves at the chiasma, by means of which the fibres from the corresponding parts of the two retinæ, carrying sensory impressions of the opposite visual field, reach the external corpus geniculatum and pulvinar of the same side, and thence the occipital cortex on the same side. That is to say, the visual impressions received on the left halves of the two retinæ are superimposed, both being received by the same part of the occipital cortex of the left side, while the visual impressions received by the right halves of the two retinæ are similarly superimposed upon the right occipital cortex. A proof of the existence of stereoscopic vision in the cat is afforded by the production of homonymous hemianopia in this animal by experimental injury of the optic tract, as I shall describe later. It is worthy of note that semi-decussation of the optic nerves is a partial return to the primitive condition in which each optic nerve arises from the same side of the brain, as seen in the cyclostoma, and in the early stage of development of the mammalian eye.

MACULAR VISION.

According to Lindsay Johnson (12) a macula is only to be found in man and the true monkeys, which, he says, alone possess the power of convergence. He states: "The divergence of the optic axes follows the classification to a marked degree. The higher the order the nearer the axes approach parallel vision. Parallel vision with the power of convergence only occurs in those animals which possess a true macula, viz., man and all the simiæ." Although this author recognises that periscopic vision (divergent optic axes, as in the hare and rabbit), combined with a large cornea, is a great source of protection to the animals by the increased range of vision it gives them, yet he does not see the obvious complement to this argument, that binocular vision, with less and less divergence of the optic axes, is of increasing value to predatory animals, birds as well as mammals, which have to follow instantly with their eyes the dodging movements of their prey in the endeavour to escape their pursuer. I have no doubt the macular region of the retina becomes more and more specialised in animals with increasingly binocular stereoscopic vision, and that the felidæ have no area of intense vision for fixation corresponding to the human macula I cannot accept on ophthalmoscopic examination alone, as Johnson asserts.

As regards the cortical representation of the macular region, there are two conflicting theories. The fact that, in most patients suffering from permanent hemianopia, the dividing line between the blind and the seeing halves of the field is found to pass around the fixation point, leaving it included in the seeing half of the field for each eye, has led Wilbrand (18) to suggest that there is a special bifurcation of the macular fibres at the chiasma, by which means the whole of each macula is represented in the occipital cortex on each side. He admits, however, that in many cases of hemianopia the dividing line passes directly through the fixation point, and he explains these and others in which it passes closer than usual to the fixation point, by suggesting that they are to be accounted for by individual variations in the amount of bifurcation of the macular fibres. Wilbrand and Saenger (19) quote, in support of this theory, the observations of Ramon y Cajal on cat embryos, who, using the Golgi method, has described fibres in the chiasma bifurcating, the divisions passing into the two optic tracts, though there is no evidence that these bifurcating fibres were macular fibres. Since Cajal has also described similar bifurcation of fibres in rabbit (4), the probability of these fibres being macular fibres is very small. On the other hand, Förster (7) regards the representation of the macula on the cortex as on the same plan as the rest of the retina, so that in a total destruction of one optic tract or of the optic radiations or occipital cortex on one side, permanent hemianopia would result with the dividing line between the seeing and the blind fields passing through the fixation point. He explains the retention of a small area of vision around the fixation point in most cases of hemianopia by supposing the macular area of the cortex to have greater resisting power to embolic or thrombotic softening through better blood supply through anastomotic branches. Against this theory is the statement that in cases of destruction of one optic tract the dividing line has been found to pass around the fixation point, leaving it in the seeing half. Henschen (11), although supporting Wilbrand's theory of bifurcation of the macular fibres, considers that Förster's supposition as to the greater resistant and recuperative power of the macular area through better blood supply as very probable. Three pieces of evidence, which, in my opinion, prove the truth of Förster's theory are (1) the mode of improvement of cases of hemianopia, especially of cases of transient hemianopia not due to any gross lesion of the half-vision centre around the calcarine fissure, but to exhaustion of the centre from the spread of an epileptiform discharge from a neighbouring patch of softening, such as occurred in the two cases which I published (9) of softening in the quadrate lobule, just in front of the cuneus. In such cases the visual field invariably clears up from the centre towards the periphery, the macular vision being the first to be regained, showing the greater recuperative power of the cortex associated with the representation of macular vision; (2) cases of double hemianopia have been recorded due to thrombotic softening in both cunei, in which permanent blindness did not result, but a small area of central vision returned, with diminished visual acuity, resembling in size the pin-point fields of hysteria.

Such recovery of central vision is a strong argument in support of the theory that the cortical area of representation of the macula has greater recuperative or resistant power than the rest of the half-vision centre. (3) The most convincing proof of the macular region being represented in the cortex on the same plan as the rest of the retina, namely, that half of each macula is represented in each cortex, is afforded by the study of cases of transient hemianopia. In the paper referred to above, I published eleven such studies of transient hemianopia occurring in four patients after epileptiform fits, in each of which the hemianopia was absolute for several hours, a bright flame being invisible in the blind field, which in each case extended up to the fixation point, the dividing line passing through the fixation point, so that the flame of a match or candle was not seen until it crossed that line. I have since then observed three similar cases, and in each vision began to clear up after several hours, until after twenty-four hours or a little longer the visual fields were again normal, the improvement taking place always from the centre towards the periphery. Moreover, every now and then one finds a case of permanent complete hemianopia in which the dividing line passes through the fixation point, and I have now under observation such a case in which a small flame is not seen at all to the right by the right eye until it crosses the line of fixation, though the left eye, which has only the smaller nasal field blind, sees the flame about five degrees to the right of the fixation line. This point has repeatedly been tested and confirmed by two other observers. To test this point it is not sufficiently accurate to use a perimeter, because it is then always uncertain whether the patient's eye does not waver a little. To make certain of the line of fixation, it is far more accurate to test these cases by Gowers' method (9), in which the patient sits or stands opposite the observer, each gazing into the pupil of the eye opposite, so that the line of fixation of observer and patient is the same, and the slightest wavering of the patient's eye is at once detected, and the test again commenced. Using this method in cases of transient hemianopia, I have in every case found the dividing line to pass exactly through the fixation point, which is, in my opinion, complete proof that in all the patients examined (seven in number) there was no special bifurcation of the macular fibres.

Förster considered that the macula was represented only in the posterior part of the calcarine fissure, while Barker (1) suggests that the representation of the macular region is shared by the whole of the calcarine cortex. von Monakow (15) also considers that there is no special bifurcation of the macular fibres, and explains the persistence in most cases of hemianopia of an area of vision around the fixation point by supposing that the whole of the visual area of the cortex, occipital and angular gyrus, is connected with the macula by means of the macular fibres in the optic tract making connections with all the nerve cells in the external corpus geniculatum. This theory is, however, negatived by Henschen's clinical and pathological work on lesions of the external corpus geniculatum, in which he proved the upper half of this nucleus to correspond with the upper quadrant of the retina. An additional and conclusive fact proving the limitation of the macular area of the cortex is the occurrence of central scotomata of hemianopic type, corresponding more or less to the macular area of vision, such as Wilbrand's case, in which local lesions of the calcarine cortex have been found.

OCULAR MOVEMENTS, CONJUGATE AND INDEPENDENT.

In man and a few of the higher mammals with binocular stereoscopic vision, the movements of the eyes are perfectly co-ordinated, so that the eyes move conjugately in all directions, laterally and vertically, as well as in convergence. This is, as is well known, due to the impressions received from the maculæ of the two eyes being received as one object on the visual cortex, and thus reflexly through the motor action of the occipital cortex upon the oculo-motor lower centres, the two eyes are kept in conjugate movement fixed upon the object, so that diplopia does not result. This conjugate movement of the two eyes, by which the optic axes are kept trained upon the same object, is the result of long-continued education of the cortex and the oculo-motor apparatus, for if the ocular movements of a young infant be watched, before the child has learnt to use its eyes in fixing objects, the eyes are often seen to diverge or to squint inwards, an independent action of the eyes which is also seen in older persons when one eye has developed blindness in early infancy. This conjugate movement of the two eyes for lateral and vertical movements (not for convergence), being the result of stereoscopic vision, is therefore directly associated with semi-decussation of the optic nerves at the chiasma. I have spent a considerable time studying the movements of the eyes of different animals, especially at the Zoological Gardens, with regard to the amount of conjugate or independent movement of the eyes possessed by them. It is not an easy matter with the majority of the animals to observe both eyes so as to be sure of their movements, especially when the eyes are placed more or less laterally, as in the sheep, oxen, and other ungulata. In the rodents I have not been able to observe any definite ocular movements at all. In monkeys and in the larger cats among the carnivora it is easy to see well-marked conjugate movements of the eyes resembling those of man, and their eyes are similarly placed in the head, with good stereoscopic vision, and I have found in sections of the chiasma of a cat stained with Marchi's method, after enucleation of one eye a fortnight previously, that a large proportion of degenerated fibres were present in the optic tract of the same side (fig. 8). In bears, a group of the carnivora which is mainly frugivorous, there is not nearly such perfect binocular vision as in the cats, the eyes at rest being divergent, and occasionally distinct independent movements of the two eyes can be seen, especially in the vertical direction. Yet the majority of the movements are conjugate, laterally and vertically, and the eyes can be brought forwards more or less in convergence. Of the ungulata, I have studied the ocular movements in the horse, ox, deer, antelopes, and sheep (mouflon and thar). In all these forms the eyes are set much more laterally than in the bear, and binocular vision is necessarily defective in these animals. Attempts at convergence are seen in all of them in the effort to look at an object directly in front of them, but it is of course imperfect. There is also a certain amount of lateral conjugate movement, but independent movements of the eyes are more easily seen in the ungulata, and are more frequent than in the bear, and occur both in lateral and vertical directions. This greater independence of movement of the two eyes is associated with a less complete stereoscopic vision, the decussation of the optic nerves being more nearly total in these animals, in the horse about one-sixth of the fibres of one optic nerve being said to pass into the optic tract of the same side. In rodents, such as rabbit and hare, conjugate movements of the two eyes could serve no useful purpose, and are practically impossible, since the eyes are set almost completely laterally, with next to no power of binocular vision. In these animals the decussation of the optic nerves is practically complete. Using Marchi's method after enucleation of one eye in a rabbit a fortnight previously to staining, I found only a very few fibres here

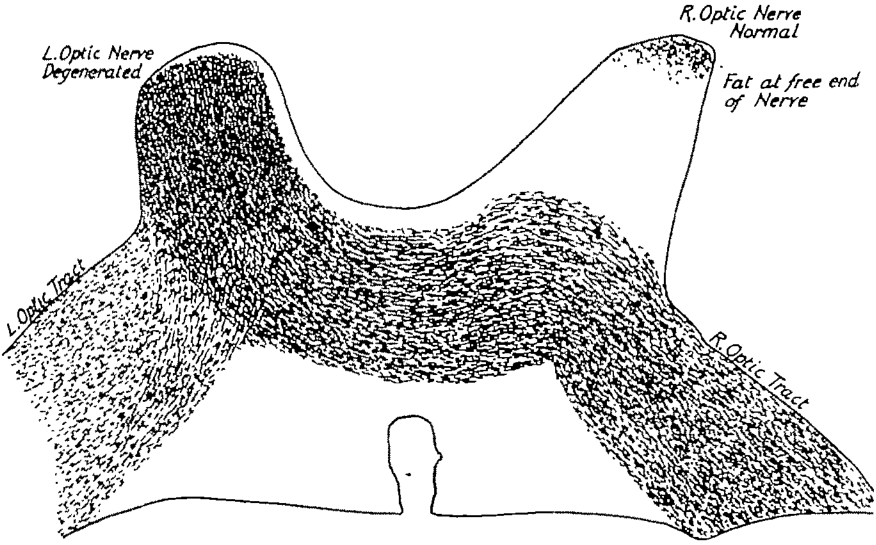

FIG. 8.

Section of chiasma of cat, stained by Marchi's method.

and there turning round from the optic nerve into the optic tract of the same side, the vast majority decussating into the opposite optic tract (fig. 7). This is also figured by Ramón y Cajal (4). As I have previously said, in all animals below mammals, whether possessed of binocular vision or not, the decussation of the optic nerves at the chiasma is complete, and they have therefore no stereoscopic vision.

Yet in many of them there is fair power of convergence of the eyes, though used solely, as far as I have been able to observe, in feeding. As I have already shown, the chameleon has the most perfect power of convergence of the eyes (see fig. 4) in the act of focussing its prey before striking with the tongue, while at all other times its eyes show the most complete independence of movement over a very wide range (see fig. 3). I have watched a large number of birds of different kinds, but in the majority it is difficult to make out any definite ocular movements, any change of direction of vision being performed by movements of the head. Especially is this the case with the owls, in none of which could I observe a trace of ocular movement. Indeed, the shape of this bird's eye is ill adapted for movements within the orbit, being somewhat egg-shaped, with a constriction round the smaller end behind the cornea. The eye is deep set, and the cornea fills the entire ocular fissure, so that very slight movements would not be easily seen. In the hornbills, however, the eyes, though much more laterally placed, are more prominent on the side of the head, and the eye movements are easily studied. The movements of the two eyes, as in the chameleon, are perfectly independent for lateral and vertical movements, though in taking food both eyes are brought forwards to bear on the object in distinct convergence. Since the eyes are, however, not so freely movable as in the chameleon, the convergence is not so perfect. The large concave-casqued hornbill at the Zoological Gardens, *Dichoceros bicornis*, makes use of this power of convergence in catching grapes or nuts thrown to it with wonderful precision.

It is thus clear that though the power of convergence is met with in animals with independent movement of the eyes and periscopic vision, yet the acquirement of conjugate movement of the eyes for lateral and vertical movements is directly dependent on the acquirement of stereoscopic vision, and the perfection of conjugate movement and the absence of independent movements of the eyes is directly in proportion to the extent of the stereoscopic vision, which is in turn dependent upon the forward setting of the eyes in the head, with parallel visual axes, and semi-decussation of the optic nerves at the chiasma. Thus the most perfect conjugate movements of the eyes, with almost complete absence of any independent movement, is seen in man and monkeys, whose eyes are set parallel, with semi-decussation of the optic nerves, and good stereoscopic vision, while complete independence of the eyes in lateral and vertical movements is seen in animals with laterally placed eyes, total decussation of the optic nerves, and periscopic vision, such as the chameleon and the hornbill. The stereoscopic vision of man, though the highest development, is not ideally perfect, since, owing to the projection of the nose, the nasal field is much smaller than the temporal, and the uncrossed bundle in the optic tract is correspondingly smaller than that which has decussated from the opposite eye.

THE PATH OF THE PUPIL REFLEX TO LIGHT.

There can be no doubt that the pupil reflex fibres for light leave the retina by the optic nerve, and run with the visual fibres in the nerve at least as far as the optic chiasma, since severance of the optic nerve, or destruction of the chiasma from the pressure of a tumour, cause abolition of the pupil reflex in addition to blindness. Occasionally exceptional cases are seen in which, although there is no perception of light from retinal disease or optic atrophy, yet the pupil may react fairly readily to light. Such are probably to be explained by supposing that the pupil reflex fibres are less easily damaged than the visual fibres. Beyond the chiasma various courses have been allotted to them. According to Henschen, the pupil fibres form a small bundle on the dorso-medial aspect of the tract. von Bechterew (2) states that they leave the chiasma posteriorly, and without decussating penetrate the floor of the third ventricle, passing uncrossed to reach the third nucleus. One of his reasons for this view is that after experimental section of the optic tract in the dog, he produced hemianopia but no alteration of the pupil. Now the dog is not nearly such a good animal for this experiment as the cat, which has a greater amount of binocular vision, keener sight, and a much more mobile pupil, reacting as briskly to light as the human pupil, while that of the dog at best reacts sluggishly. I have repeated Bechterew's experiment in cats, and have found in five animals well-marked hemianopia with the hemianopic pupil reaction in two of them, and sluggishness of the opposite pupil in all five, after injury to the optic tract on one side. Again, Darkschewitsch (5) describes the pupil reflex fibres as leaving the optic tract near the external geniculate body, and passing through the thalamus to the ganglion habenulæ and the pineal body, decussating in the posterior commissure with those of the other side, to reach the upper oculo-motor nucleus. In none of my five experiments in which the optic tract was injured did I find any degenerated fibres by Marchi's method passing into the ganglion habenulæ, or decussating in the posterior commissure; while in two other experiments in which I divided the posterior commissure, although there was temporary loss of pupil reaction (probably from shock), there was no permanent effect whatever on the pupils.

These experiments, and those necessitating the enucleation of one eye in pigeon, owl, rabbit, and cat, were performed, under the usual licences, in the Research Laboratory of Professor Vaughan Harley at University College, part of the cost being defrayed by a grant from the Scientific Grants Committee of the British Medical Association.

DESCRIPTION OF EXPERIMENTAL PROCEDURE FOR DIVIDING ONE OPTIC TRACT.

In each case the cat was anæsthetised with ether, and anæsthesia kept up until the conclusion of the operation, when, after suturing and dressing the wound with wool and collodion, the animals were kept in separate cages, and studied at intervals during three weeks. The animals were then killed, after a final examination, and the brains at once taken out and placed in Müller's fluid for microscopical examination.

Operation.—The top of the head was shaved, washed with soap, ether and turpentine, and lastly with 1 in 1,000 perchloride of mercury solution. A semicircular flap of skin was then reflected over the left ear, the temporal muscle separated from the bone and cleared away from behind forwards, and the root of the zygoma exposed and carefully cleaned. A quarter of an inch trephine hole was then made immediately above the root of the zygoma, and the hole enlarged by bone forceps upwards, forwards, and backwards, to the size of a halfpenny. The dura mater was then incised around the lower three-quarters of this opening, and the flap reflected upwards. This exposed the posterior ectosylvian fissure running vertically across the middle of the trephine opening, opposite the root of the zygoma. Through the middle of this fissure I then plunged horizontally a blunt-pointed Graefe's knife to the depth of three-quarters of an inch, cutting with the point slightly forwards. The dura was then replaced, the muscle flap returned, and the skin wound sewn up by several silk stitches, covered with boracic powder, and then with cotton wool and collodion, which was allowed to dry. The dressing was generally torn off by the animals within half an hour of their recovery from the anæsthetic, but the wounds mostly did well in spite of this.

Cat 1.—Male. Immediately after the operation the right pupil was found to be dilated and very sluggish in its reaction to a strong light held in front of it, while the left pupil reacted more briskly on exposure to light, with consensual reaction of the right pupil. On focussing the light so as to illuminate the two halves of the left retina alternately, it was found that the pupil only reacted when light was thrown on it from the left side, that is to say, the hemianopic pupil reaction could be demonstrated in the left eye. Five days later, the condition of the pupils was just as before, while there was marked right hemianopia. Some weakness of the hind limbs was apparent, and some forced movements of the trunk to the left. Three weeks after the operation the forced movements to the left had disappeared, and the hind limbs had quite recovered. The right pupil was still larger than the left, and reacted sluggishly to a bright light held directly in front, or on the left side, the left eye being shaded. No reaction of the right pupil to light thrown on it from the right side. The left pupil then reacted briskly to light thrown on it directly from the front or from the left side, with well-marked consensual contraction of the right pupil. No contraction of either pupil when light thrown on the left pupil from the right side. Both pupils contract and dilate consensually as the animal moves its eyes about, and when its attention is engaged. Examination of the animal's vision showed typical hemianopia on the right side. Each eye was in turn obscured by strapping, and it was found that the animal showed no sign of flinching or palpebral reflex to a finger darted at the eye from the right side, though on darting the finger at the left eye from the left side there was most marked flinching. Vision was certainly defective in the whole of the right eye, as was suggested by the sluggishness of the right pupil to light thrown directly on it, and the animal did not flinch at all when the finger was darted at the right eye from either direction. Yet the animal could see with this eye, for with the left eye covered with strapping, the animal could see sufficiently to find its way about, running up to the window and stopping short of the glass quite naturally, and going up to the leg of the table to try and rub off the strapping. The animal was then killed with an overdose of chloroform, and while under the anæsthetic the fundi of both eyes were examined with the ophthalmoscope, no difference in either being seen from a normal cat's fundus. The autopsy showed that the left optic tract had been wounded half an inch above the chiasma, about the anterior half of its fibres having been cut across.

Cat 2.—Medium sized male. Usual operation. Immediately afterwards the right pupil was larger than the left, and did not react directly to light, but did so consensually when light was thrown on the left pupil. The left pupil reacted briskly. The next day there was marked right hemianopia, with slight right-sided weakness and right fore-foot drop. Three weeks later there was still marked forced movement to the left. Well-marked right hemianopia, right pupil > left. No hemianopic pupil reaction could be demonstrated in this animal, though the right pupil reacted sluggishly to direct light, and more briskly consensually when the left pupil was exposed to light. At the autopsy the left optic tract was found completely cut across.

Cat 3.—Small female. Usual operation. The fissures were seen to differ somewhat from the ordinary, the posterior ecto-sylvian being short and slanting rather backwards. There were no forced movements after this operation, the animal running about quite straight an hour afterwards. There was obvious right hemianopia, the right pupil being > left, and with sluggish reaction to direct light, but reacting briskly consensually when the left pupil was exposed to light. Twenty-seven days later before killing the animal, the notes are as follows: "In good health, obvious right hemianopia, each eye tested separately, vision evidently commencing in mid line for each eye. Vision in right eye less acute than in left. Right pupil > left, and reacts sluggishly to direct light, but briskly consensually when left pupil is exposed to light. Left pupil reacts much more briskly when light is thrown on the right side of the retina than on the left side, but there is distinct reaction in the latter case also. The same, though less, for the right pupil. After killing the animal with an overdose of chloroform nine weeks after the operation and taking out the brain, the left optic tract was found wounded a quarter of an inch below the external geniculate body, but it had not been completely cut through. There was marked atrophy of the left external geniculate body and the portion of the optic tract between it and the scar, the left external geniculate body being about a quarter the size of the right one. There was no atrophy of the anterior corpus quadrigeminum, or of the pineal striæ, or of the lower part of the optic tract or chiasma.

Cat 4.—Small female. Usual operation. Immediately afterwards the right pupil was slightly larger than the left, and reacted very sluggishly to direct light, but fairly consensually. There was obvious right hemianopia, and slight right fore-foot drop. Seven weeks later the right hemianopia still persisted, the right pupil was still slightly the larger, and reacted more briskly consensually than to direct exposure to light. There was, however, no difference in the reaction to be demonstrated on focussing the light from either side on either of the pupils. At the autopsy a wound was found in the left optic tract at the neck of the external corpus geniculatum, the anterior half being divided.

Cat 5.—Medium sized female. Usual operation. The posterior ecto-sylvian fissure, which I always used as my guide for plunging in the knife to reach the tract, was in this case slightly in front of the root of the zygoma. For several hours after the operation there was marked right-sided weakness, and forced movements to the left, the animal running round after its tail. No nystagmus. Right pupil > left, with no direct reaction to light, but fair reaction consensually. The left pupil reacted fairly, and though there was obvious right hemianopia, no hemianopic pupil reaction could be made out. Nine weeks later, the hemianopia persisted, no flinching to a finger darted from the right side, though flinching at once to a finger darted at the eyes from the left side. The right pupil was very slightly > the left pupil, and reacted not quite so briskly to direct light. At the autopsy the left optic tract was found wounded at the neck of the external corpus geniculatum, the anterior half of the tract having been cut through.

Summary of Results of Wounding the Optic Tract.

Persistent hemianopia resulted in each of the cases in which the optic tract was wounded, in only one of the five cases the tract being completely divided. In each case the vision of the eye on the side opposite to that operated on was less acute than that of the eye on the same side, while the pupil reaction of the crossed eye was much more sluggish to direct light than that of the eye on the same side, and at the same time the consensual pupil contraction of the crossed eye was in each case brisker than its reaction to direct exposure to light. In only two of the five cases could the hemianopic pupil reaction be made out, in Nos. 1 and 3, three weeks and four weeks after the operation. In the first the tract was only half cut through, and in the other the tract was not quite completely cut through. No special apparatus was employed in testing for the hemianopic pupil, the light of a lamp only being focussed on the pupil from one side by means of a lens. Possibly owing to this imperfect method of confining the beam of light to one side of the retina was due my failure to obtain the hemianopic pupil reaction in every case, though it is noteworthy that in the only case, No 2, in which the tract was found to have been completely cut across, the pupils appeared to react equally well when the light was thrown from either side, although obvious hemianopia was present. Boyce (3), who divided the left optic tract in his operation for removing the hemisphere, states that this was followed by complete blindness in the right eye and slight constriction of the field in the left eye, but, as I have shown, my results do not agree with this. On examining my sections of a cat's chiasma, stained with Marchi's method (fig. 8) after enucleation of one eye three weeks previously, the number of the decussating is clearly greater than that of the uncrossed fibres. The figure of the cat's chiasma in Ramón y Cajal's work (4) shows the same outward sweep of the decussating fibres towards the root of the healthy optic nerve, as I found in my sections, and shows quite twice the number of decussating as of uncrossed fibres, which, indeed, I should have expected from the condition of the cat's vision and of its pupils in each of my five successful cases after wounding the optic tract.

It was clear from my results in these animals that the decussation of the optic nerves must be more complete in the cat than in man, less fibres turning into the tract of the same side, since the vision of the opposite eye was more affected in every case, and the pupil reaction of that eye was much more sluggish to direct exposure to light than its consensual reaction when the pupil on the same side was exposed to light. Stereoscopic vision in the cat, therefore, is less perfect than in man, in whom the vision of the two eyes in homonymous hemianopia is equally acute, and the pupil reaction to light in optic tract lesions about equally brisk in both.

In order to test the proof of Darkschewitsch's theory that the pupil reflex fibres decussate in the posterior commissure, I made four attempts to divide this commissure in cats, two of which were successful. In each case immediately after the operation the pupils were widely dilated and fixed, due, I think, largely to the greater severity of the operation. In three of the cases, however, the pupils recovered their normal movements, and in two of them at the autopsy the posterior commissure was found to have been successfully divided without injuring the corpora quadrigemina, while in another animal in which the incision missed the posterior commissure a deep cut was found in the right anterior corpus quadrigeminum close to the mid line, extending deeply to the floor of the aqueduct, and passing forwards and slightly outwards towards the inner part of the right external corpus geniculatum. In this animal, though the animal recovered sufficiently from the anæsthetic to growl, and the palpebral reflexes returned, yet the pupils remained widely dilated, with no trace of reaction to light. Unfortunately the animal died six hours later.

My operative procedure in these cases was as follows:—

Operation.—After shaving the head, and the usual antiseptic precautions, the animal being anæsthetised by ether, a longitudinal incision 2½ inches long was made in the median line from the level of the ears. After reflecting the skin and temporal muscle on the left side, I then made a small trephine opening in the parietal eminence on the left side, and then cut away the bone with forceps up to within a quarter of an inch of the median line, after changing the anæsthetic to chloroform on account of the free bleeding from the bone, which was stopped by plugging it with aseptic beeswax. The bone was then, after waiting a few minutes, cut away across the mid line and down on the right side, and was taken away posteriorly as far as the junction with the bony tentorium, leaving a hole about half an inch in length and 1½ inches in width across the middle line of the vertex. The dura was next incised along the posterior margin of the opening on the left side and reflected forwards, the posterior portion of the left hemisphere then being retracted to the left by an aneurysm needle inserted under the dura. A canaliculus knife was then passed downwards vertically in the middle line, the notch in the bony tentorium being the guide to the middle line, and being passed down to a distance previously measured on the knife as calculated to just pass the posterior commissure. The knife was drawn forwards with a cutting motion for about half an inch, the posterior margin of the corpus callosum thus being incised for about one-third of an inch, as well as the posterior commissure, which lies more deeply beneath it. The wound was then sewn up with separate silk ligatures dressed in the usual way, and the animal allowed to recover.

Of four attempts I made in this way to divide the posterior commissure only two were successful, without injuring the corpus quadrigeminum. In all the cases there was complete loss of pupil reaction to light immediately after the operation, remaining in one case for as long as three days, though the pupils still contracted and dilated consensually with the movements of the eyes. In all the animals which survived, however, the pupil reactions returned, becoming quite normal, including the two successful cases in which at the autopsy the posterior commissure was found cut, with no wound in the anterior corpus quadrigeminum of either side.

Although it appears that this operation is a greater shock to the pupil-reflex centres than is the division of one optic tract, yet the recovery of the pupil reactions to light after division of the posterior commissure disposes of Darkschewitsch's theory of the pupil fibres entering the ganglion habenulæ and pineal body, and decussating in the posterior commissure. It is indeed practically certain that the pupil reflex fibres, probably the fine nerve fibres contained in the optic nerve, reach nerve cells in the superficial grey matter of the anterior corpus quadrigeminum. On tracing the degenerated nerve fibres by Marchi's method in those cats in which I had successfully injured the optic tract, these fibres were found to surround the external geniculate body, and then by the anterior brachium to spread out over the surface of the anterior corpus quadrigeminum, none of the fibres passing into the ganglion habenulæ or into the posterior commissure (fig. 9). Indeed, it was obvious that the fibres reaching the corpus quadrigeminum by the anterior brachium must end in the grey matter of this body. Whether any of these degenerated fibres are pupil reflex fibres it is impossible to say. Probably the majority of them are fibres subserving a reflex function connecting vision with the ocular movements, especially the reflex, by means of which the eyes are held steady in position when the two eyes are directed attentively towards an object. It is possible that the fibres concerned in the pupil reflex to light running in the optic nerves do not degenerate centrally after division of the nerve or tract, if it is true that these fibres are the fine nerve fibres running peripherally to the retina, having their cells of origin in the corpus quadrigeminum, as Ramón y Cayal described.

In four of my animals (cats), in which I accidentally injured the anterior corpus quadrigeminum, I was able to trace degenerated fibres starting from the central grey matter surrounding the aqueduct immediately beneath the wound in the anterior corpus quadrigeminum, and sweeping round ventrally beneath the third nerve nucleus and the posterior longitudinal bundle, the majority decussating in the raphé and then turning caudally to form a tract running longitudinally to end in the anterior columnar fibres of the cord, as Boyce described. This is the tract named by Forel the fountain-like tegmental decussation of Meynert. This was the only degeneration I found resulting from these localised

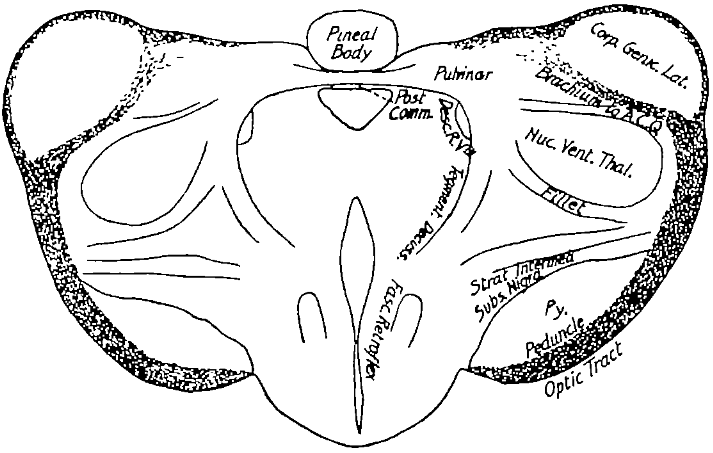

FIG. 9.

Section at level of posterior commissure of cat, stained by Marchi's method, after enucleation of left eye.

lesions of the outer and dorsal portion of the anterior corpus quadrigeminum, and it seems to me an attractive theory, as I stated in my paper before the Neurological Society in December, 1899, that these fibres are the relay continuing the path for the pupil reflex to light to the third nuclei, which has been interrupted by the nerve cells in the superficial grey matter of the corpus quadrigeminum. Not all the fibres in Meynert's fountain decussation in the cat decussate, as Boyce stated, and as I showed in my sections also, a very few running caudally in the posterior longitudinal bundle of the same side, and it seems not improbable, as Held suggested, that these fibres assist in connecting the visual reflex with all the oculo-motor nuclei and with the lower centres in the medulla and neck for turning the head.[1]

I have studied the pupil reflex to light in a considerable number of animals, frog, toad, pigeon, parrot, owl, rabbit, horse, dog, cat, monkey, and in man, and I have found that in all those animals in which there is a total decussation of the optic nerves at the chiasma, only that pupil contracts which is exposed to light. The toad's pupil is oval horizontally, and reacts very sluggishly to light, but still very definite contraction takes place, and only in the pupil exposed to the light. It is said that in these animals the iris is directly sensitive to light, but in the higher animals this is certainly not so. I have cut sections of the chiasma of a toad, stained with Weigert-Pal, and found complete decussation of the optic nerve fibres. I have also cut similar sections of the chiasma of the chameleon, and found the same total decussation of the fibres (fig. 5), but the pupil of the chameleon is too small and deeply set for any observation of its reflex to light. Considerable care is necessary in studying the pupil reflex in birds, as they possess voluntary power of constricting the pupil, in addition to its reflex contractility, the sphincter iridis in birds containing striped muscular fibres, and being unaffected by atropine. This voluntary constriction of the pupil I have noticed more in the grey African parrot than in pigeons or owls. In the parrot or pigeon, owing to the lateral position of the eyes, it is impossible to watch both pupils at the same moment, and I therefore adopted the method of fixing a mirror near one side of the bird's head, and watching the reflexion of the pupil on that side in the mirror, while throwing a beam of light upon the other pupil. After several trials, the bird becomes accustomed to the flashing of the light, and no longer contracts its pupils voluntarily at the moment of the flash. This voluntary sphincter action of the pupil is consensual, both pupils contracting equally, while the reflex contraction of the pupil to light is unilateral, only the pupil on which light is thrown contracting. The same unilateral contraction of the pupil is found in owls, although they have considerable binocular vision, and the decussation of the optic nerves in the owl, as in the pigeon, is total. I have traced the degenerated fibres in the pigeon, a fortnight after enucleation of one eye, and then cutting sections of the chiasma and optic lobes, stained by Marchi's method. The decussation is then seen to be total, all the degenerated fibres passing into the short optic tract of the other side, passing external to the insignificant geniculate ganglion, and spreading out over the surface of the optic lobe. A small bundle, the "median optic bundle," is seen running dorsally near the posterior commissure, as described by Wallenberg, and by Boyce and Warrington. This bundle has been shown by Wallenberg and Edinger to contain centrifugal fibres which degenerate downwards from the ganglion isthmi to the retina. Possibly these are the pupil fibres. There is no direct connection between the optic fibres and the third nucleus, no degenerated fibres being traced beyond the optic lobe, which corresponds to the anterior corpus quadrigeminum and pulvinar in mammals. Since, therefore, only that pupil contracts which is exposed to light, and the decussation of the optic nerves is total, it necessarily follows that there must also be a further posterior total decussation of the pupil reflex fibres between the grey matter of the optic lobes and the sphincter centre in the third nerve nucleus. Moreover, the two sphincter centres of the two third-nerve nuclei cannot be tied together by commissural fibres. The accompanying diagram fig. (10) illustrates what must of necessity be the approximate course of the pupil reflex fibres in birds and lower animals with total decussation of the optic nerves. In some mammals, such as mice, rats, and other rodents, the optic nerve decussation is either total or practically so, as I have shown to be the case for rabbits, and I have found the

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[1] This *fontainartige Haubenkreuzung* of Meynert must be distinguished from the "superior commissure of Meynert," which lies dorsal to Gudden's commissure, beneath the third ventricle, and also from another bundle known as "Meynert's bundle," which connects the ganglion habenulæ with the opposite interpeduncular ganglion. This latter bundle is sometimes known as the *fasciculus retroflexus*, and has no connection with the pupil light reflex.

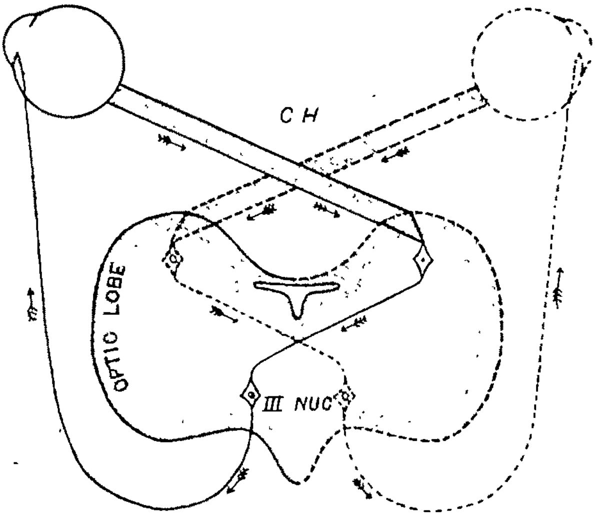

FIG. 10.

Diagram of the pupil reflex to light in birds and reptiles, and some of the lower mammals.

pupil reflex to light in rabbits to behave exactly as in the bird or toad, only that pupil contracting on which light is thrown. The rabbit's pupil is sluggish in reaction, this sluggishness probably corresponding with a poor acuity of vision. Indeed, sluggishness of pupil reaction seems to me to be associated generally with a low standard of acuteness of vision, as in ungulata and rodents, while in birds, some carnivora, as the cats, monkeys, and man, with a briskly reacting pupil to light, sharpness of vision is correspondingly more developed.

Amongst the carnivora, the cat's pupil reacts much more briskly than that of the dog, the former animal watching for its prey in dim light, which necessitates keen vision, while the latter is known to depend more upon the sense of smell than on that of sight. In the horse the pupil reacts sluggishly, as in the rabbit, and the contraction is much greater in the eye exposed to light than in the shaded one, though there does appear to be a slight amount of consensual pupil reaction, as one would expect, on account of the partial decussation of the optic nerves, one-sixth of the fibres being said to pass into the optic tract of the same side. The dog shows very fair consensual reaction of the pupils when one is exposed to light, and the same is seen in the cat. That the mechanism for the consensual contraction of the pupils is not nearly so well developed in the cat as in man is well brought out by my results on the pupil reactions after injury to the optic tract on one side. As I have already stated, after injury to the optic tract in five cases, in each case I found, although there was clearly hemianopia, yet the pupil of the opposite eye was larger, and reacted much more sluggishly on direct exposure to light than its consensual reaction when the pupil on the same side as the injury was stimulated. This indicates that the decussating optic bundle is much larger than the uncrossed bundle in the cat, as Ramon y Cajal has figured, and therefore the light stimulus, when conducted only by the small uncrossed bundle from the opposite eye, is much more feeble in causing pupil contraction than when it is conducted by the large decussating bundle from the nasal side of the retina on the same side as the injury. I have not attempted to divide the optic tract on one side in monkeys, but in all probability the results would be somewhat similar to that seen in man in cases of disease of the optic tract in which the hemianopic pupil reaction of Wernicke is seen, in which cases the briskness of reaction of the two pupils is nearly equal, whichever pupil is exposed to light. The size of the uncrossed bundle in man, and probably in monkeys, is proportionately larger than in cats, being more nearly equal in size to the decussating bundle, and its proportion is stated by Barker to vary probably from 1 : 2 to

3:5. Although human cases are on record (10) in which the whole optic nerve of one side was found to pass direct into the optic tract of the other side, yet the views of von Kölliker, though expressed so recently as 1896 (13), that the optic nerves in man, dog, cat, &c., undergo complete decussation in the chiasma, are now only of historical interest, the results of tracing degenerations by Marchi's method being absolutely conclusive to the contrary, at any rate for man and the higher mammals. Since, therefore, unilateral pupil reaction to light (birds, amphibia, rodents) is associated with total decussation of the optic nerves, so consensual pupil reaction to light is associated with partial decussation at the chiasma. In the former, in order to explain the fact that only the pupil which is exposed to light contracts, although there is total crossing of the optic nerves, there must exist a posterior total decussation of the pupil reflex fibres between

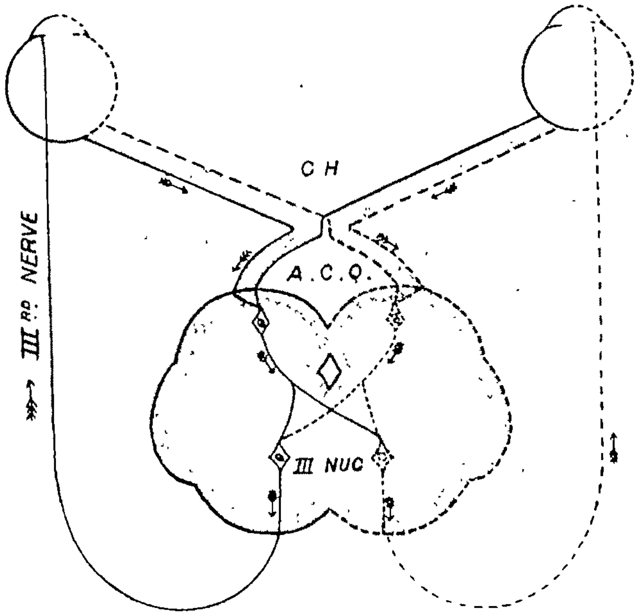

FIG. 11.

Diagram of the pupil reflex to light in man and the higher mammals.

the optic lobes and the third nuclei, which cannot be tied together by commissural fibres, at least for the two sphincter pupillæ centres (see fig. 10). To explain the consensual reaction of the pupils in man, cat, &c., it is then more consistent to suppose that the posterior decussation of the pupil reflex fibres is now partial instead of total, in the same proportion as the decussation at the chiasma has changed from total to partial crossing of the fibres (see fig. 11). This seems much more rational than to explain the consensual reaction of the two pupils by introducing a new type of fibre, viz., a commissural bundle of fibres tying together the two sphincter pupillæ centres, a commissure which we have shown to be impossible in the bird and other animals with total decussation of the optic nerves, whose pupils do not contract consensually to light, though they do so to emotional reflexes, and to accommodation. That the former is the route of the consensual pupil reflex, and not *viâ* any commissural bundle uniting the two sphincter pupillæ centres, is proved by a study of the pupil reaction in cases of uniocular Argyll-Robertson pupil.

ARGYLL-ROBERTSON PUPIL.

It is usually accepted now, on physiological grounds, that the site of the lesion in cases of reflex iridoplegia must lie between the optic tract and the third nuclei, somewhere in the interbrain, and opinions are divided as to whether the lesion is one of sclerosis of afferent fibres connecting the anterior corpora quadrigemina with the third nuclei, or whether there is an actual nuclear degeneration. Neither theory rests on pathological data or on experimental evidence. Against the former theory has been urged the fact that Argyll-Robertson pupil is sometimes accompanied by other evidences of nuclear palsy, as paralysis of accommodation, and weakness of the external ocular muscles, while against the nuclear theory may be urged the normal action of the sphincter iridis during convergence, while it does not respond to a light stimulus, and also the extreme contraction of the pupil in many cases. If the lesion were nuclear, it is difficult to understand how contraction, and not always dilatation, from paralysis of the sphincter centre, is produced.

The point that first led me to discard the nuclear theory was the results I obtained on testing cases of uniocular Argyll-Robertson pupil. I have now seen a considerable number of such cases, some in tabes, some in general paralysis, and several in patients showing signs of neither of these diseases, but in the large majority I have been able to obtain a distinct history of syphilis. If in such a case light be thrown on the affected pupil, although it does not contract itself, yet the sound pupil contracts consensually, a fact usually explained by the light impulses passing up the opposite optic tract to the third nucleus of the opposite side. Now, supposing for the moment that the lesion is in the third nucleus, if light be thrown obliquely on the temporal half of the affected pupil, as in testing for the hemianopic pupil reaction, then the impulses are confined to the uncrossed bundle in the optic tract of the same side, and reaching the diseased third nucleus, are stopped by the lesion, and neither pupil should contract, as it is inconceivable that the stimulus could pass through the lesion to reach the opposite third nucleus by the hypothetical commissural fibres binding together the two sphincter pupillæ centres without causing both pupils to contract. However, on performing this test carefully in every case of uniocular Argyll-Robertson pupil I have met with, there is equally brisk consensual contraction of the opposite pupil whichever half of the retina of the affected eye be illuminated. This seems to me conclusive against the theory which places the lesion of Argyll-Robertson pupil in the nucleus itself, if, as is usually figured in the text-books (17), one optic tract is connected through the corpus quadrigeminum only with the third nucleus of the same side. Now study of the mechanism of the pupil reflex to light in animals and birds proves the necessity for a posterior decussation of the pupil reflex fibres between the corpus quadrigeminum or optic lobes and the third nuclei, total for those animals with total decussation of the optic nerves at the chiasma, and partial for those animals with semi-decussation of the optic nerves, in proportion to the size of the uncrossed bundle. Such a path is provided by Meynert's fountain decussation which has been described above, and in my opinion some of these fibres serve the function of conveying the impulses of the light reflex from the corpus quadrigeminum to the two third nuclei of the same and of the opposite sides. Although there is no positive evidence, I believe that the lesion of Argyll-Robertson pupil is a sclerosis of these fibres, especially of their terminations in the neighbourhood of the third nerve nuclei, an hypothesis which will account for all the phenomena of reflex iridoplegia. Such a sclerosis of an afferent tract is much more in harmony with the general pathology of tabes, which is essentially a degeneration of the afferent systems of neurones, rather than a nuclear degeneration would be, nuclear lesions being comparatively uncommon in this disease. In the dilated type of Argyll-Robertson pupil there is no contraction of the pupil either to light or to accommodation or convergence, and it is probable that in these cases the lesion is now affecting also the sphincter pupillæ centre in the third nucleus. In other cases the centre for accommodation is paralysed also, with varying forms of external muscular paralysis, according to the extent of the nuclear degeneration. The typical form of Argyll-Robertson pupil is minutely contracted, due no doubt to additional sclerosis of the sympathetic dilator fibres, with some additional contracture of the muscle of the sphincter iridis due to the loss of the tonic dilator stimuli of the sympathetic.

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PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At a Meeting held at 11, Chandos Street, on Thursday, March 17, at 8.30 p.m., Dr. Alfred W. Campbell read a paper on "Histological Studies in Cerebral Localisation."

Abstract of Paper.

The aim of this work is to further the establishment of a correlation between physiological function and histological structure.

The normal topographic variations in arrangement of cortical nerve-cells and fibres have been relied upon in forming judgment on points bearing on localisation. A complete examination of a series of human and anthropoid apes' brains forms the basis of the research, and pathological material has been employed for confirmatory purposes.

The following areas have been defined, and will be briefly discussed:—

(1) *The "Precentral" or "Motor" Area.*

Confined to the precentral gyrus and the coterminous portion of the paracentral lobule; the "precentral" cortex is characterised by a great wealth of fibres and the presence of the "giant cells" of Betz. The same features distinguish the anthropoid "motor" area as defined by Sherrington and Grünbaum.

It has been found in two cases of amyotrophic lateral sclerosis that profound changes affect this area.

Differential localisation has been effected by the examination of seven hemispheres from cases of amputation, and by the discovery of *réaction à distance* (Marinesco) in the "giant cells" supposed to control muscles in the amputated member.

(2) "*Postcentral*" *Area.*

This area, readily defined in both man and the man-like ape, is limited to the postcentral gyrus and its paracentral annexe. The floor of the Rolandic fissure forms the dividing line between it and the "precentral" area, and its cortex differs absolutely from that of the latter.

Its supposed motor function is denied, and it is contended that it constitutes the terminus where fibres conveying common sensory impressions primarily impinge, on the following grounds: physiologically it is "silent" cortex (Sherrington and Grünbaum); its fibres myelinate early; it is the terminus for the "cortical lemniscus"; personal observations on three cases of tabes dorsalis have disclosed profound changes concentrated in the area.

(3) "*Intermediate Postcentral*" *Area.*

This is a skirting zone in which the structural type is intermediate between that of the "postcentral" area and the remaining parietal gyri.

(4) "*Parietal*" *Area.*

Roughly speaking this covers the superior parietal gyrus and the precuneus. It may share with the "intermediate postcentral" cortex the function of transmuting and elaborating sensory impressions primarily received in the "postcentral" cortex.

(5) "*Visuo-Sensory*" *and* "*Visuo-Psychic*" *Areas.*

The distribution of the "visuo-sensory" area is closely determined by the calcarine fissure, for the histologist its chief feature is the line of Gennari. It is believed to be designed for the primary reception of visual sensations, because its projection fibres myelinate prior to any others in the occipital lobe, and because clinicians agree that a minimal lesion in the calcarine region is equivalent to the production of a maximum of blindness.

The "visuo-psychic" area forms a wide, investing zone to the foregoing, and a remarkable wealth of fibres coupled with curious large pyramidal cells serve for its identification. It is argued that this cortex is specialised for the final elaboration and interpretation of sensations first received as crude impressions in the calcarine area.

The analogous areas in the anthropoid ape extend much more widely on the lateral surface of the hemisphere.

(6) "*Audito-sensory*" *and* "*Audito-psychic*" *Areas.*

These are the two specialised fields, definable in the temporal lobe. The first-named occupies the transverse temporal gyri. Clinical, developmental and anatomical reasons can be advanced in favour of the assumption that it constitutes the primary auditory centre and it has now been found to be the seat of grave changes in a case of deaf-mutism.

The "audito-psychic" area covers the posterior three-fifths of the first temporal gyrus and corresponds with the well-known centre for "word-deafness."

Similar areas have been found in the anthropoid brain.

The remainder of the temporal cortex, including the angular gyrus, is unspecialised.

(7) "*Olfactory*" *Area.*

Histology supports comparative anatomy in suggesting that the lobus pyriformis is the principal cortical centre governing the olfactory sense.

No explanation of the peculiar structure of the fissura hippocampi and gyrus dentatus is attempted.

(8) "*Limbic*" *Area.*

This practically covers the whole gyrus fornicatus; in the pregenual part cells are found resembling ones found in the lobus pyriformis, and it seems likely that these are end-stations for fibres of the inner olfactory root. The primitive cortical structure of the area in general belittles its functional importance.

(9) "*Intermediate Precentral*" *Area.*

This forms an investing zone on the anterior side of the "precentral" area. Embracing as it does the field of Broca and the supposed cheirographic centre, it is believed that it governs the execution of skilled movements. Its anterior boundary in the ape coincides with that of the hitherto accepted "motor" area.

(10) *"Frontal" and "Prefrontal" Areas.*

The rest of the frontal cortex is structurally divisible into two further zones of which the hinder ("frontal") is the more extensive. The feeble structural representation of the "prefrontal" cortex suggests that it is the last to appear in the course of phylogeny, and the idea is favoured that its physiological importance as a psychic centre is overestimated, but not so that of the "frontal" area.

(11) *The "Insular" Cortex.*

The "insular" cortex is all of a primitive type, that in front of the sulcus centralis has some olfactory characters, that behind temporal. It bears no resemblance to the "speech" cortex.

The paper was illustrated by lantern slides, drawings and casts.

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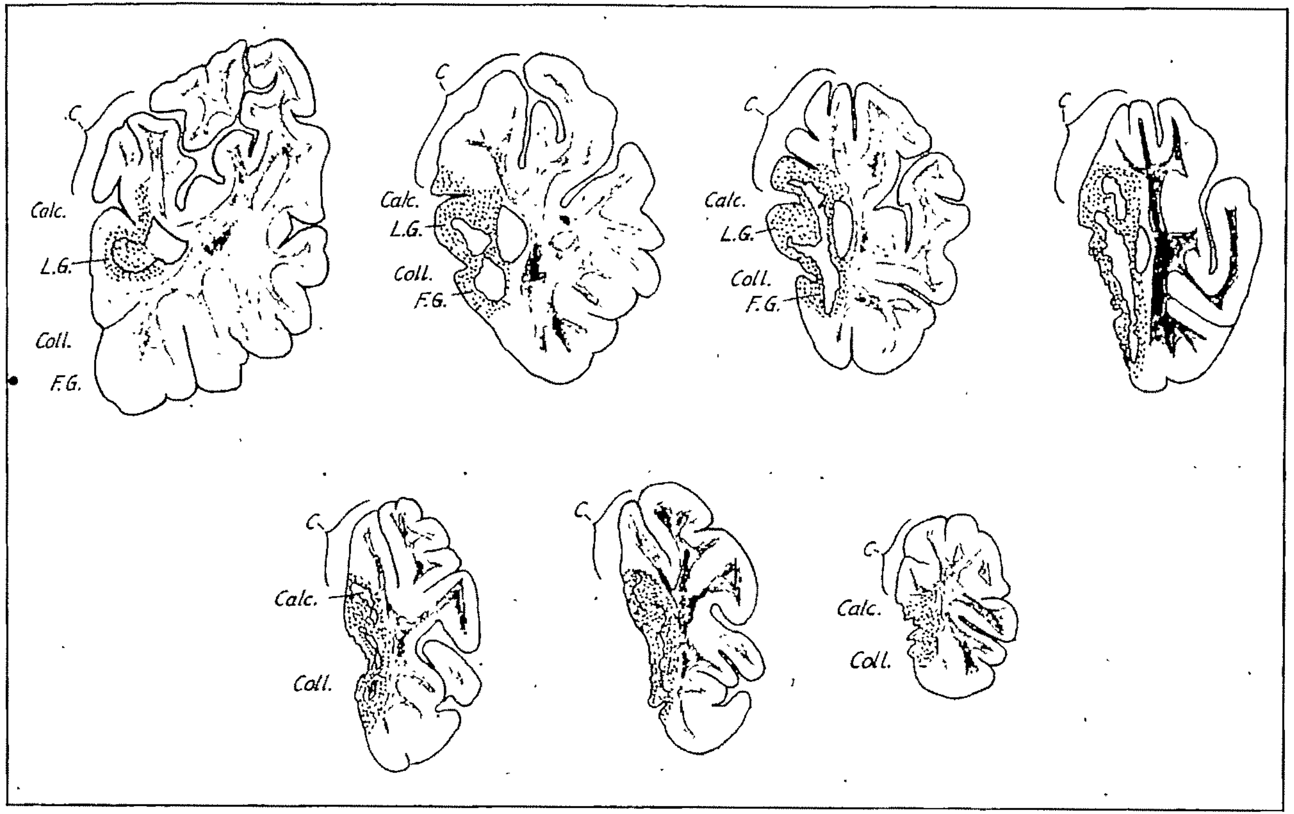
EDITOR.

cases afford evidence as to the cortical representation of the quadrants further than that the dorsal part of the visual cortex and the dorsal portion of the optic radiation between this and the external geniculate body are concerned with the lower quadrants, and that the ventral portions of both visual cortex and optic radiations are concerned with the upper quadrants. In none of these cases was the lesion confined to the cortex, and in several of them the lesion was limited to some part of the optic radiation.

The case which we report is of especial interest, since it was under our clinical observation for a long period, and careful perimetric examinations were made by several skilled observers. After the autopsy the brain was hardened in formalin, and subsequently photographs and casts of the occipital lobes were made. The occipital lobes were cut by vertical coronal sections into slices, and the posterior surface of each slice was photographed. The slices were then further hardened in Weigert's chrome alum fluid, and serial sections were prepared by Weigert's method, and projection drawings of the sections were made. These procedures allowed of the exact determination of the limitations of the lesion. The patient during the greater part of the period that he was under our observation presented, as the sole manifestation of gross nervous disease, blindness of the left upper quadrants of both visual fields, the fixation point escaping. The condition of the visual fields remained constant during a period of nearly two years. An occlusive lesion of the right posterior calcarine artery had caused destruction of the cortex (1) of the right fusiform lobe for its posterior two centimetres, (2) of the right lingual lobe from the junction of the calcarine and parieto-occipital fissures to the pole of the hemisphere, (3) of the whole cortex in the depth of the calcarine fissure, (4) of the greater part of the inferior cuneal gyrus, small areas only at the anterior and posterior limits of this gyrus being free. The necrosis did not involve the optic radiation at any point.

The only parts of the cortex of the mesial aspect of the occipital lobe which had escaped destruction were the upper two-thirds of the cuneus and the anterior and ventral



The right occipital lobe of D.C. Projection drawings of Weigert sections.
C. = cuneus. *L.G.* = lingual gyrus. *F.G.* = fusiform gyrus. *Calc.* = calcarine fissure. *Coll.* = collateral fissure.

portion of the fusiform lobe. The lower quadrants of the visual fields were entirely unaffected, and, until it was pointed out to him, the patient was unaware of the visual defect. We submit therefore that this case affords conclusive evidence that the cortex of the upper two-thirds of the cuneus is the primary visual centre for the lower quadrants.

The primary half-vision centre is considered by von Monakow to occupy the cortex of (1) the entire lingual lobe behind the junction of the calcarine and parieto-occipital fissures, (2) the entire cuneus and extending for some half an inch or more on to the external aspect of the occipital lobe. He thinks that the calcarine fissure probably forms the line of separation of the representation of upper and lower quadrants.

It must be borne in mind in connection with Monakow's localisation of the half-vision centre that Ramón y Cajal traced the axons of the neurons of the external geniculate body to a corresponding area of the cortex in mammals. Further, the fibres of the optic radiation have been proved to form the layer of Gennari, which is a peculiarity of the cortex of the mesial aspect of the occipital lobe. Dejerine figures the layer of Gennari as having almost the same limits as von Monakow's half-vision centre. It is found throughout the cuneus and the corresponding portion of the lingualis, ceases abruptly below at the collateral fissure, and extends from the cuneus for some little distance onto the convexity of the occipital lobe. Brodmann gives much the same limits to Gennari's layer ; Bolton, on the other hand, considers that it is absent from the upper convolution of the cuneus. Henschen finds that this layer is much more marked in the cortex lining the calcarine fissure, where it is divided into two separate layers.

In the case here recorded Gennari's layer was well marked in the upper two-thirds of the cuneus on the side of the lesion, and in the left occipital lobe it had such limits as Brodmann has described. Further, we have examined the occipital lobes of several normal brains, and have found this layer constantly present both in the upper part of the cuneus and in the whole of that part of the lingual lobe which lies below the cuneus.

Monakow's conception of the limits of the half-vision centre is opposed by Henschen, who considers that, while the bulk of the cuneus and lingualis are doubtless concerned with half vision as secondary centres, the primary half-vision centre is located in the cortex of the lips and depth of the posterior calcarine fissure. The depth of the calcarine fissure is so close to the optic radiation that necrosis of the former is apt to involve the latter directly, and since in our case the whole of this cortex was destroyed and yet the lower fields were unaffected, it seems possible that direct involvement of the optic radiation is accountable for the homonymous hemianopia which has been reported in cases of necrosis confined to the region of the calcarine fissures.

We wish to draw especial attention to the following points in our case, and to submit these inferences.

(1) The larger part of the lesion was situated below the calcarine fissure. (The upper quadrant is chiefly if not entirely represented below the calcarine fissure.)

(2) The cortex lining the calcarine fissure was completely necrotic. (The primary half-vision centre cannot therefore be limited to the calcarine cortex nor the macula exclusively represented in the anterior part of this region.)

(3) The portion of the half-vision centre, as limited by von Monakow, which was not involved, was the upper two-thirds of the cuneus.

(4) The visual defect was blindness of both left and upper quadrants. (The lower quadrants are represented to a great extent in the upper two-thirds of the cuneus.)

(5) The lower quadrants of the visual fields were not affected, and the patient was not himself aware of any visual defect.

Deductions from these facts strongly support von Monakow's localisation of the half-vision centre, since the supply of the lower quadrants in our case was certainly from the upper two-thirds of the cuneus. The calcarine cortex to which Henschen limits the half-vision centre was entirely destroyed, and according to his localisation there should have been homonymous hemianopia.

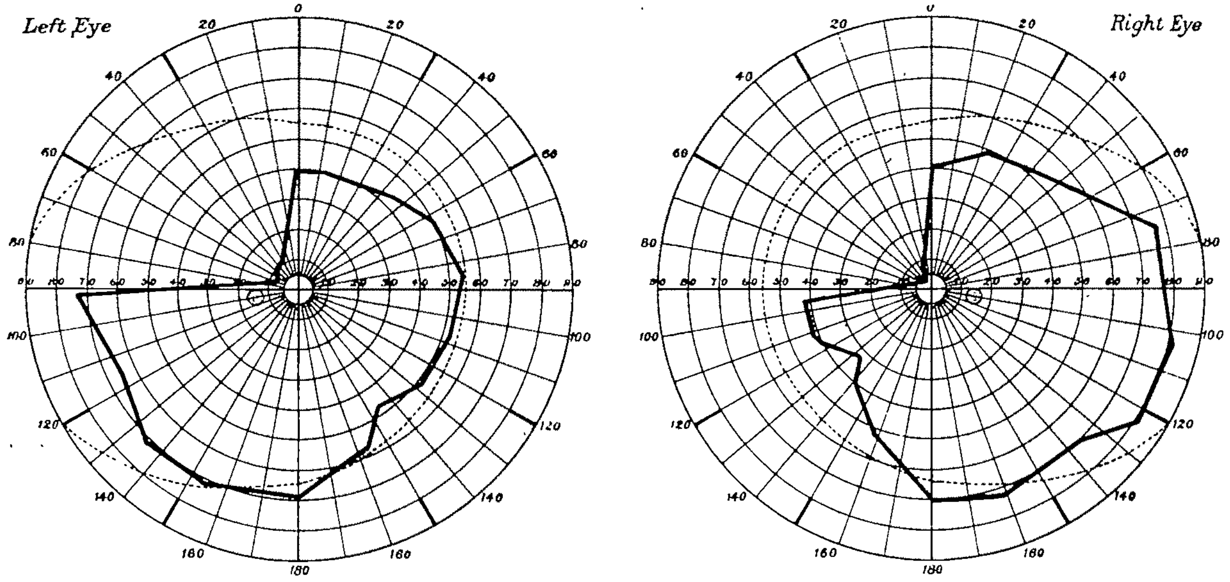


Chart of the visual fields of D.C., taken on May 25, 1900.

According to Bolton, who excludes the upper part of the cuneus and restricts the half-vision centre to a pear-shaped area tailing off in front and occupying the lingualis and inferior cuneal gyrus, there should have been hemianopia, for the limits of the lesion in our case corresponded almost precisely to the limits of his half-vision centre. There is some difficulty in reconciling our results with those found in the only other case of quadrantic hemianopia with a pathological examination, and which was published by Hun. In this case the left lower quadrants were lost and the lesion upon the surface involved the inferior cuneal gyrus alone. In our case the inferior cuneal gyrus was almost entirely necrosed and the lower quadrants were not affected.

Hun made no microscopic examination, and his case is open to the judgment which has been expressed by Bolton and many others, and which our case fully confirms, that the extent of a lesion cannot be judged from its surface appearance and that the lesion as found by microscopic examination is invariably of greater extent than it appears upon macroscopic examination. The probable explanation of Hun's case is that the necrosis of the inferior cuneal gyrus extended somewhat deeply so as to cut off the radiation of the whole cuneus. We think, further, that cases of hemianopia with the lesion confined to the calcarine cortex are to be explained on the same grounds, that the lesion involves the optic radiation.

It would seem from our case that the line of separation between the visual centres corresponding with the upper and lower quadrants is at the upper limit of the inferior cuneal gyrus, but in this connection a most important consideration arises. In the only two cases of quadrantic hemianopia which to our knowledge have been examined, the case here reported and that of Hun, there has been complete or nearly complete destruction of the inferior cuneal gyrus. In the one case the upper quadrant was lost, the lower intact; in the other case the lower quadrant was lost and the upper was intact. The limits of the quadrants in the two cases corresponded. In our case we argue that the loss of the upper quadrant was due to the upper limb lasted two days and then ceased, but sudden attacks of a similar sensation commencing in the fingers and running up the arm to the face occurred from time to time. There was no impairment of consciousness and no twitching of the muscles in these attacks.

He was never aware of any defect in the visual field.

When admitted the only abnormalities detectable were that he was blind in the upper left quadrant of the field of both eyes. The pupils reacted well to light and equally from the blind and seeing areas of the field. His vision was $\frac{6}{6}$ in either eye, and he could read J2 when he was wearing his reading glasses. There was no subjective sensory disorder. He complained at times of severe occipital headache.

There was some arterio-sclerosis and some signs of cardiac hypertrophy, but no albumen in the urine.

The optic discs were pale. The retinal arteries showed marked " silver wire " change.

There was no alteration in the appearance of the blind quadrants upon ophthalmoscopic examination as compared with that of the seeing quadrants. He was discharged in July, 1900.

He remained in much the same condition till August 18, 1901, when his sight became much worse. Details of this visual affection were not obtainable, presumably it was the result of the recent softening found *post mortem* in the region of the chiasma and right optic tract.

He became queer in his head and had three attacks of sudden unconsciousness without convulsion. Great physical feebleness came on and he was re-admitted in August, 1901. He was very restless and delirious, and difficult to examine. There was no motor or sensory paralysis. The optic discs seemed normal. The following day the respirations became grouped and he died on September 12, 1901.

PATHOLOGICAL ABSTRACT.

The convexity of the brain showed no peculiarity. There was much patchy thickening of the internal carotid arteries and basilar artery, and of their branches.

After hardening in formalin the cerebellum was removed, revealing a patch of old softening much sunk below the level of the surrounding convolutions, and occupying the mesial aspect of the right occipital lobe. A small recent patch of softening was present at about the middle of the left occipito-temporal (fusiform) convolution, while a quite recent thrombosis of fine vessels had caused some softening of the right optic tract, just behind the chiasma of some of the underlying gray matter.

Measured antero-posteriorly the right hemisphere was 4 centimetres shorter than the left. The scar on the mesial aspect of the right occipital lobe involved upon the surface:—

(1) The posterior two-thirds of the lower convolution of the cuneus, the larger upper convolution of the cuneus being entirely uninvolved.

(2) The posterior extremity of the lingual lobe for 2·5 centimetres.

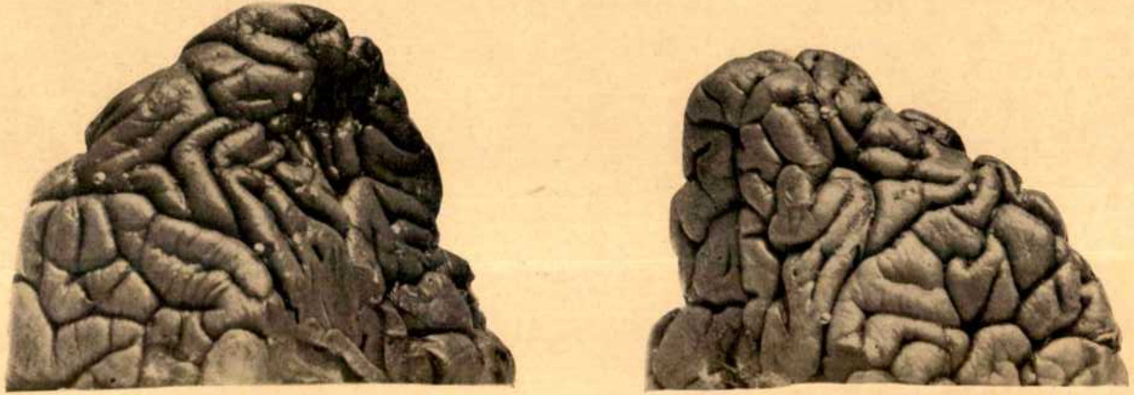
(3) The posterior extremity of the fusiform lobe for 2 centimetres.

Vertical sections through the occipital lobes were made, the photographs of which are shown on the plate. The pieces were then hardened; fluid and serial sections were prepared by the Weigert-Pal method.

These sections showed that the softening was more extensive than could be judged from the surface appearance of the lesion. It reached to and involved the inner wall of the posterior horn of the lateral ventricle, and extended in this position as far forwards as the splenium callosi, gradually tapering, and being last visible at that level as a small patch inferior and mesial to, and in close contact with, the posterior horn.

In sections through the cuneus the softening involved the lower one-half of the inner wall of the posterior horn of the ventricle. At its upper limit it involved most of the medulla of the lower convolution of the cuneus, but that of the upper convolution of the cuneus was not affected. It extended downwards as far as the inferior limit of the hemisphere in the subcortical white matter.

So far as the cuneus was concerned, the softening was entirely confined to the lower convolution.



The mesial aspects of the occipital lobes of D.C. The Chart shows the surface extent of the lesion as determined macroscopically.

At the posterior extremity of this convolution the gray matter bordering on the calcarine fissure alone was affected. For its middle two-thirds the whole of the convolution was involved, while in front of this the lesion became limited again to the lower part of the convolution, and at the junction of the calcarine and parieto-occipital fissures it involved the depths of the calcarine fissure only.

In front of the internal parieto-occipital fissure the softening extended into the lower and posterior part of the gyrus fornicatus.

The optic radiation, lying close to the outer wall of the posterior horn of the lateral ventricle, showed marked degeneration by the Weigert-Pal method. Between this region and the external geniculate body the regeneration was apparent in the ventral portion of the optic radiation.

The superior colliculus of the right side was slightly smaller than that of the left side, but it presented no other peculiarity. The mesial geniculate bodies of the two sides appeared of equal size. The right external geniculate body was smaller than the left. Very conspicuous was the difference in size and appearance between the pulvinars of the thalami upon the two sides. The posterior mesial projection of the thalamus was practically absent on the right side, and the superior brachium, markedly smaller than that of the left side, was uncovered by the pulvinar.

The right optic tract was much smaller than the left, and was soft and brown in colour from a recent local thrombosis just behind the chiasma. The right optic nerve was distinctly smaller than the left. The optic disc presented no peculiarity to the naked eye, nor on microscopic examination.

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* *This paper reviews all previous work and contains a full bibliography up to the date of its publication*, 1900.

SOME CASES OF FAMILY DISSEMINATED SCLEROSIS.

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CASES of disseminated sclerosis occurring in families are so rare that the following are worth putting on record, and they also illustrate very well the aberrant types of disseminated sclerosis often so difficult to diagnose with certainty.

THE FAMILY X.

The first case to come under my notice was John X., the fourth child, whom I saw originally in May, 1898. He was then 30 years of age, and while in India, some months before, he had noticed that he became easily tired, that then his gait became very uncertain, and finally so altered that he was sent home. When I saw him he was very ataxic in the movements of his hands, arms and legs; his gait was so uncertain that he somewhat at first sight resembled a case of tabes dorsalis, but there was a little dragging of the feet (spastic gait) and it was found that his knee-jerks were much increased, and ankle clonus was present on both sides. It was almost impossible, owing to the ataxia, for him to write, and there was slurring of the speech, but no true "scanning." The patient was peculiarly emotional, and if he was moved suddenly to laughter his head dropped forward, his sense of equilibrium failed him, and he would have dropped to the ground if he had not been at once supported. There was no nystagmus, no paralysis of eye muscles, and no optic atrophy.

Since 1898 he has, with occasional remissions, much improved; there is now but slight incoördination in walking, but if anyone present makes a humorous remark his friends always at once rush to him to prevent his falling, as the incoördination comes on with laughing as before. He has lately developed a goitre, but has no tachycardia and no fine tremors.

The second child, Mary X., aged now 37, came to see me in September, 1900, and was then suffering principally from acid dyspepsia; but she also stated that she had had three attacks of "neuralgia," starting from the right shoulder and reaching round to the right breast. She had been suffering also for three weeks from some numbness of the outside of the left thigh, and for five days in the right thigh also. She said the numbness reached almost to the waist, but that it was not a total loss of sensation, and on testing it was very difficult to make out exactly where the numbness was, as her replies were so uncertain. Knowing, as I will relate later, how neurotic the family was, I thought it likely that most of her troubles were "functional," but in September, 1902, she again came to see me, saying that the numbness of her legs was better, but that for ten days she had had numbness of the right hand, which she attributed to having carried a heavy basket a few days before. Again on examination it was impossible to say exactly where the numbness was, but I noticed a slight slurring and slowness of the speech. Three days later the paræsthesia and partial numbness had extended over the whole right side of the body, and she had become irritable in temper. In July, 1903, the last time I saw her, the peculiar sensory disturbances had reached to the head and were still on the right side of the body and right limbs; there was great uncertainty of movement with the right hand, the "excursions" in unnecessary directions being marked; she could only hold a pen with difficulty and her handwriting was very sprawling and infantile; there was slight atrophy of the thenar muscles of the right hand. No other symptoms or signs of any kind were present, and the eyes were normal in all respects. Since that date I hear she has been "better and worse," but she has developed nothing further.

The sixth child, Edward X., now aged 30, came to see me on January 24, 1902. For three years before this time he had much work and worry, and suffered from constipation, but he was fairly well until the hot weather in the summer of 1901, when on several occasions, while playing tennis or cricket, he suddenly lost control of the left leg, and fell down. At the end of November, 1901, he noticed slight numbness of the fingers of the left hand, especially in the thumb and index finger. A few days before his visit to me he noticed that the sight of the right eye was blurred, and I found he could not distinguish a face at the distance of four feet. He also told me that in 1895 he had double vision, which lasted for two months. There was no nystagmus; the right optic disc was very indistinct, probably from a descending neuritis; his speech was very slow and slurring, but not scanning. He has never had syphilis. 1 have not had another opportunity of examining this patient, but I am informed by his doctor that he has improved very much, except that the speech is still slow.

The rest of the family history is somewhat remarkable.

The father, aged 66, was strong and healthy up to about eighteen months ago, when, after a monetary loss, he became somewhat depressed, and is now suffering from melancholia, with delusions of poverty and unworthiness, and is, probably, suicidal. A first cousin (male) of the father died some years ago from advanced disseminated sclerosis, and a cousin of this man (a girl) came to see me a few weeks ago suffering from very marked exophthalmic goitre.

The sister of the mother of the X. family came to see me in 1900, when she was 52 years of age. She was suffering from insomnia and melancholia, with ideas of unworthiness and an inability " to find comfort in her religion."

The other children of the family X. have been three in number. The eldest was a female, who died in an asylum while suffering from melancholia. The second is Mary, mentioned above; the third is a female, who suffers periodically from severe sciatica; the fourth is John, mentioned above; the fifth is a male, now living abroad, and said to be well; the sixth is Edward, mentioned above.

Although on paper it may seem that the signs and symptoms manifested by the three children suffering from disseminated sclerosis are slight, yet to the patients themselves they are very disabling, and especially so in the case of John. They are in a good social position, and their many engagements are much interfered with by their disease. It may possibly be thought from my description that a diagnosis of disseminated sclerosis is not justified in all the cases; but the symptoms are, on personal examination, much more pronounced than can be easily described. It is so difficult to exactly pen what the ear detects in the altered speech and what the eye sees in the slightly unsteady movements when definite actions are performed. At least two of the cases have been seen also by eminent London neurologists, who have at once confirmed the diagnoses.

The Family Y.

Miss Jane Y., aged 25, was brought to see me by Dr. Sellers, of Rochdale, on April 12, 1904. About ten years ago she suffered from pains in the head and eyes. About five years ago she had double vision when looking to the right, but this only lasted a few months. Four years ago she commenced to walk badly, was unsteady in her gait, and occasionally stumbled; rarely, her legs suddenly gave way, and she fell to the ground. She was seen about this time by a well-known and highly-experienced neurologist, who could find no real indications of organic disease, and thought it probable that the symptoms were functional. Since that time she has had remissions and relapses; for a time she had numbness of the left side of the body.

When I saw her the gait was very slightly spastic in character, and when turning round suddenly there was a little uncertainty of movement. There was no sign of actual paralysis, but some uncertainty in using the hands. The knee-jerks were increased, but there was no ankle clonus. She said she had occasional slight numbness of the right index finger, but no definite alteration of sensation could be found on examination. Sometimes the handwriting is bad, and her speech is very slightly slurring, thick, and nasal, and her father said that at times he could hardly understand what she said. There is no nystagmus, no paralysis of eye muscles, and the optic discs are normal.

The family Y. consists of three daughters—Mabel, aged 29; Cissy, aged 27; and Jane (mentioned above), aged 25. Cissy is in good health, but Mabel is suffering from advanced disseminated sclerosis, as will be seen from the following account, kindly supplied to me by Dr. Sellers. In the early part of the year 1900 she had been walking badly, and sometimes fell. In August, 1900, she had an attack of influenza, after which she suddenly "went stiff all over." In October, 1900, she was seen by a physician, who diagnosed lateral sclerosis, and said she would only live six months. She became quite an invalid, and had to be carried downstairs. In April, 1901, she improved somewhat, was able to be dressed and sit out of doors, and in May was able even to walk upstairs. In November, 1901, she became worse, had to stay in bed, and could not move her left leg. Since that time she has gradually got worse, and now (March, 1904) she is scarcely able to walk even by holding on to a chair. Her right arm is tremulous, and she cannot hold anything with it; her speech is thick and scanning. There is no nystagmus.

The father tells me that Jane is "just like" Mabel was at the beginning of the latter's illness.

COMMENTARY.

Heredity.—As I have already stated, it is rare to find instances of disseminated sclerosis running in families. Sir Wm. Gowers says ("Diseases of Nervous System," vol. ii., p. 544): "Direct heredity, or the affection of two brothers or sisters, has been noted in a few instances, but is quite exceptional; indirect inheritance is more common, shown by a family history of insanity, epilepsy, or of some form of chronic paralysis." Dr. Risien Russell, in his excellent account of the disease ("Allbutt's System of Medicine," vol. vii., p. 51), says: "It has only happened exceptionally that any direct hereditary connection has been traced, or that more than one member of the family has been affected;" and he mentions two observations by Leuch and Eichhorst in both of which a mother and child were affected. I am inclined to think, however, that the more we become familiar with the aberrant types of disseminated sclerosis, the more it may be found to occur in families; that is to say, where one member of a family has typical disseminated sclerosis, another member who has been diagnosed to be merely "neurotic" may really be found to be suffering from aberrant disseminated sclerosis. The family X. also illustrates well the connection of the disease with mental diseases in the same family.

Type of disease.—It will be noticed that most of the cases reported are suffering from the irregular or aberrant type of disseminated sclerosis, a condition which ought to be better known and studied, for these cases are frequently diagnosed as hysteria, with lamentable results, especially for the diagnostician. It is unnecessary here to give a lengthy account of aberrant disseminated sclerosis, as such an account is so easily accessible in Dr. Risien Russell's article referred to above; but it may not be out of place to epitomise the points illustrated by my cases, and which enabled me to make my diagnoses. These are: Irregular subjective patches of numbness, often varying, disappearing and reappearing; sudden falls from giving way of a leg; slight paralysis of hands, lasting for months or years; transitory paralyses of eye muscles; optic atrophy; subjective feelings of giddiness; alteration of handwriting; sudden dropping of articles held in the hand; ataxic movements of hands, and sometimes of feet; spastic condition of legs, with true ankle clonus, Babinski's toe extension reflex (if any reflex is obtained at all[1]); alteration of speech, even very slight (a sign which I consider of the utmost value). In the early stage the speech may not be "scanning," but may be a "slur" not unlike that found in early paralysis of the insane, or it may be a slight difficulty in forming certain words.

Prognosis.—Not only from observing for long periods many cases of aberrant disseminated sclerosis, but also from observations on the fully-developed disease (especially in the Manchester Workhouse Infirmary, where cases remain for many years), I am of opinion that the hopeless prognosis so often given in these cases is hardly justifiable. I do not mean that disseminated sclerosis recovers, but I do think that it may last for many years, and that even when fully developed very great improvement may occur, so that a patient who has been bed-ridden may be able to walk about again and be comparatively comfortable. Moreover, the symptoms of the aberrant type may almost disappear (as in the case of John X. and Edward X.); at any rate they may last for many years without passing into the fully-developed disease. I think it is important that this should be more recognised, as one can truthfully instil hope and give encouragement to these sufferers.

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[1] This reflex was not tested for in the X. family. Jane Y. did not respond to sole irritation.

Disseminated Sclerosis and Exophthalmic Goître.—Last year a young lady was brought to see me who was suffering from disseminated sclerosis and from exophthalmic goître, both fully developed. It will also be noticed that a cousin of the X. family suffers from exophthalmic goître, and that John X. has developed a slight goître. These cases may be, and probably are, the merest coincidences, but in such diseases as disseminated sclerosis and exophthalmic goître, in which the etiology is so obscure, the coincidence seems to me to be worth bearing in mind.

FÆCAL VOMITING AND REVERSED PERISTALSIS IN FUNCTIONAL NERVOUS (CEREBRAL) DISEASE: A SUMMARY OF CASES AND CONCLUSIONS.

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SOME of the older literature bearing on the question of reversed peristalsis in the alimentary canal has been collected by J. J. Woodward.[1] A. G. Guainierio[2] gave the story of a man who vomited a suppository introduced into his rectum. J. Matthias de Gradibus[3] related the case of a girl, aged 12, who not only vomited fæcal matters, but afterwards enemata, which were ejected by the mouth shortly after they were introduced into the anus. This state of things lasted three days, during which suppositories introduced into the rectum were vomited in the same manner; when it was attempted to keep a suppository in place by a string, the string broke, and it was speedily vomited. D. S. Sennert cited a case observed by J. Optheus, in which a piece of tallow candle, used as a suppository, was vomited shortly after its introduction. Diemerbroeck recorded a case in which a suppository was vomited, and likewise a case in which an enema was ejected by the mouth. Morgagni has referred to a number of similar cases.

The following case I shall narrate in some detail, as I had an opportunity of observing the patient herself during some part of the original illness[1]; moreover, she was temporarily under my care about six years afterwards for somewhat similar, but less severe, symptoms.

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[1] "Medical and Surgical History of the War of the Rebellion," Washington, 1879. Part II., vol. i., p. 836.

[2] *Tractatus de Fluxibus*, Cap. 2, Lyons edition of 1534, fol. 136.

[3] *Practica, de Aegritudinibus Stomachi*, Cap. 5, *de Vomitu*, fol. 213, Venice edition, 1502.

Rosa S., aged 22 years, a fairly healthy-looking young woman, was admitted into the German Hospital, October 19, 1896. According to the history given, the patient had been quite healthy up till the time of her marriage in 1896. On the night of her marriage her husband had hæmoptysis[2] and died not long afterwards. From this time the commencement of her symptoms must apparently be dated.

The chief of these symptoms were the following:—Vomiting (sometimes with a little blood), distension of the abdomen, and great constipation. There was no evidence that the idea of possible pregnancy had anything to do with the distension of the abdomen, which was afterwards found to disappear at once under chloroform. The vomiting persisted on and off. At one time there was undoubtedly fæcal vomiting. Actual scybala or formed fæces from the large intestine were certainly vomited on more than one occasion. At times, when an oil enema was administered, some of the oil reappeared in the vomited matter. In order to guard against imposture and to obtain an accurate diagnosis, an enema coloured with methylene blue was administered by the nurse. Some of the methylene blue appeared in the vomited matter within ten minutes after the administration of the enema. Any deception on the patient's part was altogether impossible.

This observation and the history of hæmatemesis seemed to furnish reasonable grounds for supposing that a fistulous communication between the colon and stomach or duodenum might exist. It may be noted, however, that when the gastric contents were evacuated by the syphon tube immediately after an enema containing methylene blue had been administered, no methylene blue was found to be present in the contents of the stomach. This showed that the methylene blue injected by the rectum took at least some minutes to reach the stomach. To make a long story short, it may be stated at once that during the patient's residence in the German Hospital two careful exploratory laparotomies were performed. At neither of them, however, was any abnormal condition whatever detected. One of the laparotomies was performed below the umbilicus, the other above. At the second operation the stomach itself was opened and explored. On both occasions the wounds healed rapidly and no unsatisfactory results occurred. There may, indeed, have been some temporary improvement in the general condition following the operation. The improvement, however, was certainly not permanent, and the patient's condition varied considerably at different times.

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[1] Vide *St. Bartholomew's Hospital Reports*, 1898, vol. xxxiv., p. 315, where, by the kindness of my colleagues, Dr. Port and Dr. Michels, I was allowed to give notes of the case.

[2] This part of the history was, as I subsequently ascertained, inaccurate in my original account. I have therefore corrected it here.

During the patient's residence in the German Hospital her temperature was found every now and then to be above the normal, and this appeared to be especially the case when she was not being closely observed. It was not, however, proved that she simulated the fever by manipulating the thermometer. Before the patient left the hospital (February 4, 1897) she looked well, had a fresh colour, and moved about quickly, but was still troubled with vomiting, constipation, and distension of the abdomen.

On February 5, 1897, she came under the care of Sir F. Treves at the London Hospital. Her symptoms there were much the same as at the German Hospital, and so convinced was he that some local disease might be present that he determined to do a third laparotomy. This was actually performed, and, though the exploratory operation gave a negative result, no evil consequences resulted, but on the contrary, the patient's condition seemed to be improved after the operation. Treves has alluded to the case in his paper on "Abdominal Section as a Medical Measure," read at the Medical Society of London on February 28, 1898.[1] The portion of his paper relating to the patient's progress after admission to the London Hospital I will now quote at length, since it amply confirms some of the points alluded to above:—

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[1] *Transactions of the Medical Society*, vol. xxi., p. 224.

"Shortly after admission she exhibited definite hysterical attacks. She had, by some means, acquired the power of causing the mercury in the clinical thermometer to rise to the limits of the instrument. One medical man who had attended her wrote to say he had recorded a temperature of 110° F. No action of the bowels could be obtained. She would howl with pain for hours. All food taken by the mouth was vomited; nutrient enemata were given by the rectum, but they also were vomited. A careful investigation of this vomiting of enemata was carried out by my house surgeon, Dr. Scars, with the aid of the sister of the ward. An enema of castor oil was given; within ten minutes from the time of the introduction of this drug into the rectum the whole of the castor oil, as demonstrated by actual measurement, was vomited from the mouth, together with a small scybalous mass. A few days later, in order to further test this phenomenon, an enema of one pint of water stained a deep colour by methylene blue was injected into the rectum by the sister in the presence of the house surgeon. The whole of this enema, to the amount, that is, of one pint, was vomited by the mouth in ten minutes. I was extremely ill-disposed to carry out a third abdominal section. The only excuse for it was that, while at the previous operations the stomach had been carefully examined, an equally detailed examination had not been made of the colon. As the patient resisted all forms of treatment, vomited all she took by the mouth, vomited nutrient enemata, and had no action of the bowels, and as she was becoming somewhat alarmingly feeble, I resolved once more to carry out an abdominal section as a forlorn hope. The abdomen was opened in the left semilunar line above the level of the umbilicus. The rectum and the whole length of the colon were examined with the greatest care and minuteness, and found to be absolutely normal. Some few adhesions existed around the scar of the wound in the stomach, but, with this exception, the abdominal cavity did not exhibit any trace of disease. The patient thought fit to be very ill after the operation, her respirations at one time reaching 140; she could not be induced to speak, and she went through all the popular phenomena of dying with startling effect. As these death-bed displays were not encouraged, she took finally to screaming, and became so intolerable in the ward that she was removed to an isolated room. The absence of an appreciative audience appeared to have an immediate effect upon her symptoms, for she soon ceased to complain, the bowels acted without difficulty, the vomiting ceased, the temperature remained normal, and before she left the hospital, on March 19th, she may be said to have been perfectly restored to health. The highest temperature she was able to develop while in the hospital was 109°. She had stated that she could produce this heroic fever by very slowly squeezing the bulb of the thermometer between her teeth. An attempt to produce this elevation of the mercury by the means indicated only led, however, to the destruction of two thermometers."

After leaving the London Hospital the patient again attended the out-patient department of the German Hospital, where, in September, 1898, I saw her again whilst doing the out-patient work for my colleague, Dr. Michels, during his temporary absence. There was no longer any vomiting, but a marked tendency to flatulent distension of the abdomen still persisted ("hysterical tympanites").

In December, 1902, the patient was temporarily under my care at the German Hospital for hysterical vomiting and hysterical tympanites. She at that time showed right hemianæsthesia (of which she was not aware until it was discovered by medical examination), hysterical contraction of the visual fields, and absence of pharyngeal reflex. As the abdomen and lower part of the thorax showed no respiratory movements the tympanites seemed to be due to tonic contraction of the diaphragm combined with relaxation of the other abdominal muscles.[1] In regard to the fever, which was present according to the chart, there was almost certainly an attempt on the patient's part to deceive us. On December 22 a thermometer was broken in taking the morning temperature and a second thermometer registered 102° F. After this endeavours were made to prevent any tampering with the thermometers, and from that date till the patient left the hospital on January 12, 1903, her temperature never reached 100° F.

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[1] S. Talma (*Berliner klin. Wochenschr.*, February 3, 1902, p. 90) maintains that "hysterical tympanites" is produced, when the tympanites is general, by tonic contraction of the diaphragm combined with relaxation of the other abdominal muscles, but when the tympanites is localised (forming one kind of "phantom tumour"), by relaxation of some and contraction of other muscles. This hysterical tympanites may be removed by an anæsthetic, but when the effects of the anæsthetic commence to pass off, the diaphragm ceases to ascend properly with each expiration, and thus gradually the peculiar tympanitic condition reappears.

It would be a mistake to suppose that extreme symptoms of this kind are confined to the female sex, for this type of hysterical disorder is likewise found in men, just as most other forms of hysteria are. The following case,[1] from Professor Senator's clinique in Berlin, presents certain features in common with those of the preceding case.

The patient, A. H., was a shoemaker, aged 29. He was tolerably healthy up to the time of his military service in 1890. In this year, by some accident, he fractured one of his left ribs. Whilst in the hospital for this injury he suffered from obstinate constipation, which persisted, more or less, from that time. When he recovered from the fractured rib he was freed from liability to military service, and then visited a number of hospitals on account of his constipation. In 1893 he is said to have had fæcal vomiting, and in 1894 was supposed to have intestinal stenosis. Besides persistent vomiting, severe constipation and distension of the bowels, with pain in the lower left region of the abdomen, patient also suffered from polyuria resembling diabetes insipidus. About the end of the year 1894 an exploratory laparotomy was performed with negative results. The patient's condition, however, was temporarily improved after the operation. Later on, he had obstinate constipation for fourteen days and a second laparotomy was performed. A fibrous band was removed from the peritoneum, but the surgeon thought that the presence of this band had probably nothing to do with the symptoms. The patient's condition again improved after the second operation, and for some time he could again do his work. However, in November, 1897, he was troubled with his old symptoms. In January, 1898, the patient was admitted into the Charité Hospital at Berlin, with obstinate constipation, continual vomiting and distension of the abdomen. The bowels were made to act and the vomiting was relieved by energetic treatment, namely, lavage of the stomach, enemata, and purgatives. Morphium, to which the patient had already become accustomed, was administered on account of the abdominal pains, &c. In spite of the treatment a motion of the bowels was only obtained about every four or five days. Every two or three weeks there was an ileus-like attack, with persistent vomiting and absence of any action of the bowels, not even the passage of flatus. Lavage of the stomach relieved these acute exacerbations. There was no genuine fæcal vomiting. The urine was increased in quantity, of rather low specific gravity and of pale colour, without albumen or sugar, and generally without much indican. At times there was retention of the urine, lasting twenty-four hours, when the bladder would reach nearly up to the umbilicus. This retention of urine was sometimes relieved by a hot bath, but sometimes the use of a catheter was necessary. Other occasional symptoms were dyspnœa, migraine, hiccoughing, &c. On May 18, 1898, there was a typical hysterical attack, and then the great distension of the abdomen suddenly disappeared. In the evening the bowels were opened spontaneously for the first time. After this the patient's condition was much improved; he could get about again, and his abdomen felt flaccid; purgatives, however, were still usually necessary.

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[1] *Vide* Strauss, "Hysteria Virilis unter dem Bilde der Chronischen Darmstenose. Zweimalige Laparotomie." *Berliner Klinische Wochenschrift* 1898, No. 38, p. 838.

Gilles de la Tourette[1] has collected a number of cases presenting analogous symptoms, including several examples of the vomiting of enemeta.

In a hysterical woman, aged 27, Briquet[2] found that an enema of coffee was partially returned by the mouth fifteen minutes after its introduction per rectum. A coloured substance was injected into the rectum to settle all doubts on the question, and twelve minutes afterwards some of it appeared in the vomit. The investigation was so arranged in this case that no deception was possible.

Jaccoud[3] records the case of a hysterical woman who suffered for some days from vomiting of fæces. Every precaution was taken to avoid the possibility of deception. Though the patient did not seem to be gravely ill, this extraordinary vomiting, which Jaccoud terms "buccal defæcation," continued until the eighth day, when the bowels began to act again in the natural manner. A few weeks afterwards this patient died of typhoid fever, and at the necropsy nothing was discovered to explain the fæcal vomiting. The ileo-cæcal valve appeared natural in every respect.

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[1] *Hystérie Paroxystique*, Paris, 1895, vol. ii., p. 361.
[2] Quoted by Gilles de la Tourette, *loc. cit.*, p. 363.
[3] Quoted by Gilles de la Tourette, *loc. cit.*, p. 363.

Tullio[1], in his paper on "Reversed Peristalsis," records a case of this kind, where the intestinal movements could be observed through the abdominal walls. The movements were noted to start from the region of the sigmoid flexure, and to pass along the colon into the small intestine. A lump then began to form in the pyloric region, and continued to increase until, the pyloric sphincter doubtless opening, vomiting took place. These intestinal movements were increased when an enema was administered, and ceased after it was vomited.

I now come to H. Schloffer's[2] excellent paper on the subject of hysterical ileus, and shall quote from an abstract I made at the time for a medical journal. According to Schloffer, Professor S. S. Rosenstein[3] noticed fæcal vomiting in a healthy-looking boy, aged 9, following attacks resembling hystero-epilepsy. The fæcal masses which were vomited on one occasion were coloured blue by some henna, which had been administered in the form of an enema. Under bromide treatment the boy recovered after several months. In this remarkable case the boy, when apparently in perfect health, would suddenly have a convulsive seizure, with severe opisthotonos, and on the attack coming to an end he would throw up formed fæces by the mouth almost at the same time as he passed an ordinary motion of similar formed fæces by the anus. Cherchevsky[4] has given the case of a nervous constipated man, aged 42, considered to be suffering from a spastic neurosis of the bowel; during one such attack a formed piece of fæces, eight centimetres long, was vomited. For a short time after this the patient was faint, but one and a half hours later his bowels acted easily and copiously. In J. B. Murphy's[1] case the patient was a man, aged 40, who had previously suffered from attacks of lead colic; a laparotomy was performed on account of persistent constipation and signs of intestinal obstruction. A piece of intestine, eight inches long, was found contracted like a solid cord (see the illustration in Murphy's paper), and during the operation was seen to relax. Within three hours after the operation there was an action of the bowels. W. von Leube[2] observed fæcal vomiting in a girl, aged 19 years, and a hard cord could be felt in the abdomen along the whole colon from the cæcum to the sigmoid flexure. This could no longer be felt twenty-four hours later on, but ten days afterwards a similar attack occurred. P. Sandoz[3] has noted a case of tabes dorsalis where hiccough and ileus followed a gastro-intestinal crisis and lasted during days, and then disappeared after free action of the bowels. L. Heidenhain[4] thinks that worms, &c., may cause reflex spasm of the intestines, and thus give rise to symptoms of intestinal obstruction. In one case a laparotomy was performed, but no obstruction could be found. A round worm, however, could be felt in the small intestine (which was otherwise empty and collapsed or contracted). Several doses of morphine were given, and the bowels acted three and a half days after the laparotomy. A. Voisin[5] records that a hysterical girl twice recovered from the symptoms of intestinal obstruction under antispasmodic and purgative treatment, but died during a third attack, and the necropsy pointed to mere spasm of the bowel. According to A. J. Hoorweg, a woman, aged 26, who had already had severe hysterical attacks, suffered from fæcal vomiting, anæsthesia, psychical disturbance, &c. On washing out the stomach in the morning, remains of the evening meal were found, and in the evening the stomach often contained fæcal masses. Cousot[1] has published a case of fæcal vomiting following long constipation in an hysterical woman with hemianæsthia and concentric contraction of the fields of vision. Slajmer performed a laparotomy in the case of an anæmic hysterical woman, aged 26, with fæcal vomiting and symptoms of intestinal obstruction. Nothing abnormal was found except that a portion of the small intestine was firmly contracted. This contracted portion became relaxed during the operation. Next day the bowels acted. Similar intestinal symptoms later on could often be relieved by simple antihysterical treatment. A married woman, aged 28, who had previously had hysterical symptoms, as well as hæmoptysis and hæmaturia, without definite signs of organic disease in the lungs or urinary organs, was operated on on account of fæcal vomiting. The results of the laparotomy were negative, excepting that portions of the small intestine were found firmly contracted. The patient, however, was given to understand that the operation had been successful. Five days later the bowels acted after enemata, and the patient recovered. Wölfler operated on a woman, aged 31, with fæcal vomiting and symptoms of chronic intestinal obstruction. The small intestine was found to be closely contracted at certain spots between portions containing scybalous masses. A few days after the laparotomy normal motions occurred, but the patient still had often to employ laxative drugs.

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[1] Quoted by Gilles de la Tourette, *loc. cit.*, p. 866.

[2] "Ueber Ileus bei Hysterie," *Beiträge zur klin. Chirurgie*, Tübingen, 1899, vol. xxiv., p. 392. Schloffer's article, a carefully-written critical summary of literature on the subject, should be consulted in the original by those interested, not only for the conclusions he arrives at, but also for the numerous references he gives.

[3] *Berliner klin. Wochenschr.*, August 21, 1882, p. 521.

[4] "Contribution à la Pathologie des Névroses Intestinales," *Révue de Médecine*, Paris, 1883, p. 896.

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[1] *Journal of the American Medical Association*, January 4, 1896, p. 20.

[2] "Ueber Ileus Spasticus," 70 *Versammlung deut. Naturforscher und Aerzte zu Düsseldorf*, 1898, Zweiter Theil, II. Hälfte, p. 64.

[3] *Correspondensblatt für Schweizer Aerzte*, Basel, 1887, p. 41.

[4] "Beiträge zur Pathologie und Therapie des acuten Darmverschlusses," *Arch. für klin. Chirurgie*, Berlin, vol. 55, p. 211, and vol. 57, p. 1.

[5] Referred to in the *Gazette Médicale de Paris*, 1876, No. 51, p. 609.

Schloffer also quotes the case recorded by Desnos of a man who fell from a wall against some scaffolding, striking his abdomen. After the accident apparently he had no normal action of the bowels, but every day, at about six o'clock in the evening, he got rid of his fæces by the mouth. The man seems to have been hysterical, and Schloffer suggests it may have been a case of hysterical fæcal vomiting, but further evidence would be desirable in regard to such a very extraordinary case.

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[1] *Neurol. Centralblatt*, Leipzig, 1893, p. 50 (abstract from *Bulletin de la Soc. de Médecine Mentale de Belgique*, September, 1892).

I will now go on to the papers by M. Sander[1] and G. Langmann,[2] again making use of abstracts which I wrote at the time for a medical journal. Sander records the case of a hysterical man, aged 23, with neuropathic family history. In December, 1894, about two months after commencing his military service, he began to suffer from attacks of abdominal pain, meteorism, and unconsciousness, with epileptiform movements. The pulse, when observed in May, 1895, was found to vary in the course of twenty-four hours between 60 and 100. There was diarrhœa alternating with constipation. In November, 1896, the patient was in the Frankfurt Hospital, with variable abdominal symptoms and occasional evening fever. He was supposed to have appendicitis, and laparotomy was performed. The vermiform appendix was found slightly injected and removed. Soon after the operation there were again attacks of meteorism and pain, with evening fever to 101·6° F. Improvement followed in December, 1896, but there was an attack of eructation, salivation, grinding of the teeth, and twitchings of the limbs. In January, 1897, there was again meteorism, with abdominal pain (chiefly in the umbilical region) and occasional vomiting. Sometimes there was fever, and on one day itching of the whole body. There was generally constipation, and blood and mucus were noted in the motions. On account of a suspicion of intestinal obstruction a second laparotomy was performed on January 14, 1897. On January 29 the patient jumped through the window and ran about barefoot in the snow, so that he had to be removed to the lunatic asylum. In the asylum, amongst other troubles, he had much vomiting, and in consequence lost strength, but suddenly, on February 19, 1897, he began to improve in every way, although a certain tendency to vomit persisted. Not long after leaving the asylum he was reported to have (apparently not seriously) attempted suicide.

A second case, reported by Sander, is that of a hysterical woman, aged 24, whose father had been addicted to alcohol.

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[1] *Deut. Med. Wochenschrift*, 1899, No. 36.

[2] *Festschrift A. Jacobi*, New York, 1900.

She had had hysterical trouble of the right hip joint, factitious erythema, &c., before she suffered from abdominal pain, vomiting and apparent fever in 1895. At different times gastric ulcer, appendicitis, and peritonitis were thought of. From September, 1897, to September, 1898, laparotomy was performed on four occasions with negative results. She was then transferred to the lunatic asylum, where her psychical condition commenced to improve. There was a certain amount of right-sided anæsthesia.

Langmann[1] describes the case of a young woman, aged 21, who was admitted to the German Hospital in New York, in 1889, for vomiting and hæmatemesis. Four years previously the left leg had been apparently paralysed (hysterical paralysis?) for nine months after an injury. Since that time severe pain along the spine (hysterical rachialgia?) was occasionally felt. During the two years before admission the patient was said to have had attacks of peritonitis, vomiting, and hæmatemesis, and had habituated herself to hypodermics of morphine. In the hospital the patient sometimes vomited fæcaloid material, sometimes actual hard formed fæces. Scybala, an inch thick, were at one time thrown up. A fistulous communication between the stomach and colon was suspected, owing to repeated hæmatemesis and circumscribed pain in the gastric region. Indigo administered with an enema was ejected, together with fæces, from the mouth in less than fifteen minutes. An exploratory laparotomy was performed, but nothing abnormal could be discovered except a darning needle, which was impacted in the anterior wall of the stomach between the serous and mucous coats. The wound healed well, and the vomiting ceased. About thirty-three days after the operation, however, nausea with vomiting of mucus and blood recommenced. Afterwards, hard fæces were brought up again. About six weeks after the operation the stomach was washed out, and tepid water containing indigo was injected into the rectum. The patient was carefully watched, so that deception could be excluded, and yet nine minutes after the enema was administered she vomited a fæculent mixture of milk coagula with indigo. A week later some hard fæces wrapped in paper were found under the patient's pillow. The patient left the hospital soon afterwards, and was detected in some tricks at another hospital. She was then lost sight of. Langmann believes that the stercoraceous vomiting was genuine and spontaneous, and could not be brought on by will, and that only after perceiving on what point the medical interest centred did she try to imitate a symptom which had created so much sensation.

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[1] *Loc. cit.*

L. E. Bregman[1] reported the case of a servant girl, aged 23, admitted to the hospital with symptoms of intestinal obstruction. There was a history of absolute constipation for ten days. She had vomiting and tympanites, with abdominal pain, but under an anæsthetic the tympanites disappeared. The vomiting was the most prominent symptom. There was sometimes ordinary vomiting after food, sometimes hæmatemesis, sometimes fæcal vomiting. Bregman, who was often present during the attacks, was able to exclude trickery. The fæcal vomit had the colour and odour of fæces and contained solid fæcal particles, or occasionally larger fæcal masses. Intestinal irrigation sometimes brought away fragments of fæces or scybala, but often the water was returned merely coloured. The symptoms varied in severity from time to time, but finally there was general improvement. The case was, however, complicated by prolapse of the rectum, which was treated by operation. Headache, backache, amenorrhœa and dysuria were complained of, and there were some other troubles, such as attacks of aphonia. The tendon reflexes were increased and the conjunctival and pharangeal reflexes diminished.

Debove[2] gives the case of a Jewish patient, aged 36, who had symptoms of complete intestinal obstruction, with tympanites, severe abdominal pains and retention of urine, following an attack of influenza with great constipation. Though the symptoms were alarming, yet the general condition remained relatively good, and ultimately the bowels acted spontaneously and fæces with an enormous quantity of gas were discharged. No evidence of any organic disease could be found in the patient. A valuable point in favour of delaying operation in this case was the definite history furnished by the patient's brother (a medical practitioner) of the patient having previously had similar attacks of various degrees of severity which had not necessitated operation. Mental excitement, chill, and certain articles of food seemed to induce an attack.

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[1] "Ueber Kothbrechen bei Hysterie," *Neurologisches Centralblatt*, Leipzig, October 1, 1901, page 882.

[2] "Pseudo-étranglement interne d'origine névropathique," *Presse Médicale*, Paris, November 22, 1902, p. 1119.

Sander's and Debove's cases, though not examples of fæcal vomiting, serve capitally to illustrate the subject of neurotic intestinal obstruction, that is to say, of symptoms resembling those of intestinal obstruction, occurring in functional nervous cases. In this place I shall not enter further into the literature of such cases nor of the majority of cases (with or without neurotic vomiting, but without any fæcal vomiting) which have been recorded as "hysterical pseudo-ileus," "hysterical spurious gastric ulcer," "hysterical pseudo-appendicitis," or "hysterical pseudo-peritonitis," many of which cases have led to exploratory laparotomies. At present I am concerned with the subject of (neurotic) actual fæcal vomiting, and from a study of the cases to which I have referred it is possible to obtain an idea of the general sequence of events in this rare symptom-complex, which I shall subsequently point out is intimately allied to ordinary attacks of hysterical vomiting.

Typical history of an attack of hysterical fæcal vomiting. The patient, generally a young woman, but sometimes a man or a child (of either sex), may seemingly be in blooming health before the attack. Some distressing mental emotion or other physical or psychical shock is followed by a period of great constipation, and this constipation is accompanied or followed by meteorism (hysterical tympanites) and abdominal pains. There may be severe attacks of vomiting and even some hæmatemesis. The constipation becomes absolute and the other symptoms get worse, and finally the condition of "hysterical ileus" is reached. Then everything taken by the mouth is returned. The vomit becomes fæcal in character and even pieces of formed fæces may be ejected by the mouth. Enemata and suppositories may likewise be vomited. Hysterical symptoms, such as hemianæsthesia and concentric contraction of the visual fields, are likely to be found if sought for, and there may be occasional hysterical fits or attacks of hystero-epilepsy. The ejection of fæces by the mouth in one case (Rosenstein's) used to follow convulsive seizures resembling hystero-epilepsy; in another case (Senator's) a typical hysterical attack preceded the disappearance of the abdominal distension, &c. The onset of the attack may sometimes be more acute than I suppose it to be in typical cases. Once started the symptoms may continue for weeks or months, with irregular remissions or intermissions, and then gradually or rapidly subside, either spontaneously or under bromides, strict isolation and antihysterical treatment; an exploratory laparotomy perhaps sometimes, but certainly not always, exerts a slightly favourable influence by suggestion. Recurrence after a longer or shorter interval (which, however, may not be quite free from minor hysterical troubles) is to be expected, and other severe hysterical conditions, including the mental state leading to "hysterical malingering," are not unlikely at some time or other to show themselves.

Evidence of Reversed Intestinal Peristalsis (Antiperistalsis) and of the Passage of Fæces backwards through the Ileo-cæcal Valve.

Many experiments have been made and a good deal has been written on this subject. Dr. Arnott[1] said long ago:—"From an erroneous opinion that what has been called the valve of the cæcum acts as a perfect valve, allowing passage downwards only, few practitioners have ventured to order much liquid to be injected for fear of overstretching or bursting the lower part of the intestines; and the possibility of relieving disease above the supposed valve has scarcely been contemplated. It is now ascertained, however, that fluid may be safely injected, even until it reaches the stomach." Oskar Kraus,[1] who has carefully studied the structure of the human ileo-cæcal valve, comes to the following conclusions:—that the valve is mechanically imperfect in new-born children, but becomes a perfect valve in early life; that the valve can be rendered incompetent by excessive distension of the colon; that in cases of insufficiency of the valve the insufficiency may be either due to persistence of an infantile condition or may have been acquired in later life; there is a possibility that the nervous system may have some influence in regard to the opening of the valve. In dogs and cats,[2] and other lower animals, the ileo-cæcal valve is rather a muscular sphincter, like the pylorus, than a purely mechanical valve, and therefore, as Kraus points out, experiments on animals are not of much value for investigating the action and value of the mechanical valve found in human beings.

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[1] Quoted by Woodward (*loc. cit.*) from J. Scott, *Commentaries on the Use and Necessity of Lavements, &c.*, London, 1829.

It is possible that in cases of fæcal vomiting the backward passage of the intestinal contents through the ileo-cæcal valve may be due (1) to acquired incompetency resulting from forcible distension of the colon, or (2) it may be due to congenital incompetency, the valve having retained its infantile type (like the muscular sphincter of lower animals), or (3) it may be due to opening of the valve by muscular action connected with antiperistaltic movements in the colon. On the one hand, Dr. A. E. Maylard[3] refers to a boy who died of peritonitis and who vomited enemata before his death; linseed oil injected per rectum appeared on the surface of the material vomited. The necropsy showed absence of a proper ileo-cæcal valve, and the preparation by William Hunter is preserved in the Hunterian Museum of Glasgow.

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[1] "Noch einmal: Zur Anatomie der Ileocöcalklappe," *Wiener klin. Wochenschrift*, 1902, No. 19. See also Max Herz, "Ueber die Insufficienz der Darmklappe," *Wiener Ges. für inn. Med.*, February, 1902. Kraus points out that the structure of the normal human ileo-cæcal valve and its perfect valvular action were accurately described in the anatomical lectures of Archangelus Piccolomini, printed in 1586.

[2] T. R. Elliott (*Journal of Physiology*, May, 1904), comes to the conclusion that the junction of the small and large intestine in cats is controlled by a muscular sphincter, not by a mechanical valve.

[3] *British Medical Journal*, December 31, 1892, p. 1451.

On the other hand, in Jaccoud's case, already alluded to, the post-mortem examination is said to have shown a perfectly normal ileo-cæcal valve. Whatever may be the exact explanation, it is certain that, in cases of fæcal vomiting, the contents of the large intestine do somehow pass backwards through the valve.

It is also certain, in spite of what surgical text-books say, that the most typical cases of fæcal vomiting are due, not to organic obstruction in the large intestine, but to intestinal contraction and antiperistalsis of nervous origin. Leichenstern and Nothnagel regard the presence of formed fæces in the vomit as one of the greatest rarities in cases of ordinary intestinal obstruction, and Schloffer[1] thinks that the vomiting of formed fæces would rather point to a case being one of functional nervous origin. In the so-called "fæcal vomiting" of organic obstruction the vomited material is generally only fæculent, that is to say, there is only sufficient admixture of fæces to give the vomit a smell of fæces.

Nor is this surprising if one pictures to oneself what probably takes place. When there is organic obstruction in the large intestine the gut above the obstruction is likely to become distended and the ileo-cæcal valve insufficient,[2] then it needs merely ordinary contraction of the gut and of the abdominal walls to make the contents of the small intestine fæculent if not actually fæcal; formed fæces, however, are unlikely to be passed back through the ileo-cæcal valve in such cases.

Violent antiperistaltic movements are probably required before pieces of formed fæces can be passed up from the colon through the ileo-cæcal valve, and violent movements are more likely to occur in a gut with normal muscular walls than in a gut weakened by disease and over-distension; in fact, violent antiperistalsis sufficient to produce the passage of formed fæces through the ileo-cæcal valve is more likely to be due to nervous derangement than to organic disease or obstruction of the gut. The experimental evidence as to the occurrence of antiperistalsis is somewhat conflicting, but Nothnagel[1] showed that coloured salt solution injected per rectum could be carried by antiperistalsis through the ileo-cæcal valve into the small intestine, and Grützner[2] thought that small particles of charcoal, when injected as an enema suspended in salt solution, could under favourable circumstances reach the stomach within a few hours. Grützner's results have, however, been called in question. W. B. Cannon,[3] who studied the movements of the intestines in animals by the help of bismuth food and Röntgen rays, found that part of the enemata (containing bismuth for purposes of radioscopy) passed the ileo-cæcal valve apparently simply owing to antiperistalsis in the colon. He never saw "food material pass back from the colon so far as the stomach; but once, about ten minutes after an injection of 100 cc. of warm water, the cat retched and vomited a clear fluid resembling mixed water and mucus. In the fluid were two intestinal worms still alive." Dr. A. G. Jacobs[4] obtained evidence in two girls who were being fed by nutrient enemata (one on account of severe hæmatemesis, the other on account of persistent vomiting in early pulmonary tuberculosis) that at one time some of the material used for the enemata must have found its way by antiperistalsis to the stomach. Jacobs goes further and explains the possibility of complete absorption of large enemata by supposing that part of the enemata is carried upwards into the small intestine, and that absorption is thereby greatly facilitated. R. Saundby[5] says that when he was administrator of anæsthetics at the General Hospital, Birmingham, a very emaciated youth had his thigh amputated at the hip-joint for disease. The operation was rather long, and he vomited up bile-stained mucus for some time. After disarticulation, a large nutrient enema of hot milk and brandy was given, the last part of the operation being suspended for the purpose. The patient afterwards began to throw up uncurdled milk, evidently derived from the enema.

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[1] *Loc. cit.*

[2] For support of Haguenot's "overflow" theory of fæcal vomiting, see Prutz and Ellinger, "Ueber die Folgen der Darmgegenschaltung," *Arch. für klin. Chirurgie*, Berlin, 1904, vol. lxxii. In this connection it may be pointed out that a localised intestinal paralysis could probably (as Heidenhain and Schloffer have suggested), give rise to the symptoms of obstruction ("ileus paralyticus"), just like a localised intestinal spasm can. This might explain the symptoms of intestinal obstruction noted in some cases of embolism of mesenteric arteries, the sudden arterial ischæmia leading to a state of "ischæmic cramp" or "ischæmic paralysis" (followed by actual necrosis) of the portion of gut dependent for adequate blood-supply on the obstructed artery.

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[1] *Beiträge zur Phys. und Path. des Darms*, Berlin, 1884.
[2] *Deut. Med. Wochenschr.*, 1894, No. 48.
[3] *American Journal of Physiology*, 1902.
[4] Ueber Rectalernährung," *Festschrift Julius Lazarus*, Berlin, 1899, p. 185.
[5] *British Medical Journal*, 1892, vol. ii., p. 1140.

I think it is quite clear that the upward passage of fæces in genuine cases of nervous fæcal vomiting is due to active antiperistalsis. Even in ordinary severe vomiting there may doubtless be a certain amount of antiperistalsis in the upper portion of the small intestine, as the vomit is frequently greenish from the presence of bile.[1] In some cases of simple and bilious vomiting during exclusive rectal feeding, such as H. D. Rolleston and Jex-Blake[2] recorded in 27 per cent. of ninety-six gastric ulcer cases, the vomiting may probably be regarded as the equivalent of antiperistalsis in the stomach (with or without actual antiperistalsis in the duodenum), excited in sympathy with the antiperistalsis in the colon, which, according to the experiments of W. B. Cannon,[3] seems to be normally set up by the introduction of nutrient enemata into the rectum.

Diagnosis.

In the diagnosis of cases of fæcal vomiting due to functional nervous disease there are several points for consideration. The presence of a *fistulous gastro-colic communication* may be at first suspected, and such a suspicion may be strengthened by the previous history of abdominal pains and hæmatemesis, a history which is not rarely obtained in cases of severe functional nervous vomiting. Indeed, laparotomy has been performed in several cases of nervous fæcal vomiting owing to the symptoms being thought to point to the presence of a gastro-colic fistula. H. D. Rolleston,[1] however, referring to these two affections, points out some important differences in the symptoms. He refers to the writings of Bec[2] and Bouveret[3] on gastro-colic fistula. According to Bec's collection of sixty-two cases, a fistulous communication between the stomach and colon is usually due to cancer or ulcer of the stomach, and Bouveret and Tournier believe that when gastric carcinoma is the cause, the carcinoma is generally, if not exclusively, one which has developed in an old ulcer. Bec's sixty-two cases show that among the chief symptoms of gastro-colic fistula are fæcal vomiting, vomiting of enemata, abundant diarrhœa, fæcal odour of the breath, thirst, and rapid wasting. Whereas free diarrhœa is generally a marked feature in cases of fæcal vomiting due to gastro-colic fistula, in the functional nervous cases constipation is the general rule. Moreover, cases of the functional nervous class are unlikely to be accompanied by such rapid wasting and anæmia as are characteristic of true gastro-colic fistula.

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[1] The presence, however, of bile in the vomited matter, when there is repeated vomiting from any cause, may also merely be due to temporary slight relaxation of the pyloric sphincter and to suction action of the stomach dependent on the vomiting movements.

[2] "On the Occurrence of Vomiting during Rectal Alimentation," *British Medical Journal*, 1903, vol. ii., p. 68.

[3] *Loc. cit.*

In *fæcal vomiting due to organic obstruction* of the bowel (organic ileus) there is of course constipation, but the other symptoms differ considerably from those characteristic of functional nervous cases. In the latter cases the general aspect of the patient is not nearly as bad as one would expect, whereas the extreme gravity of the typical surgical cases is quite apparent. Moreover, as has been already explained, the vomit in organic obstruction can generally only be recognised as fæcal or fæculent by its odour, whereas in some of the functional nervous cases actual formed fæces, evidently from the large intestine, may be thrown up.

In the functional nervous cases *symptoms of hysteria*, such as hemianæsthesia, concentric contraction of the visual fields, absence of pharyngeal reflex, hysterical aphonia, hysterical palsies, hysterical local spasms or hysterical general convulsions, may aid the diagnosis.

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[1] *Practitioner*, London, August, 1899, p. 199.
[2] *Thèse de Lyon*, 1897.
[3] *Révue de Médecine*, Paris, April 10, 1899, p. 324.

In regard to *pain* as an aid to diagnosis I believe that, in spite of the work of Head and others on pain and hyperæsthesia in visceral disorders, pain is still often misleading in cases with abdominal symptoms. Very many persons have doubtless undergone dangerous and useless operations owing to their constantly complaining of pain incorrectly supposed to be due to some organic disease.

The vomit in cases of *organic nervous diseases* may occasionally, though rarely, be more or less fæcal or fæculent in character. In the gastric crises of tabes dorsalis the vomit not rarely contains bile from the duodenum, but in pure tabes it hardly ever, if ever, becomes fæculent. In the case of a tabetic man recorded by Paviot,[1] after a period of constipation and persistent vomiting, the matter thrown up from the stomach was at last fæculent. A laparotomy was performed, but the intestines appeared not to be diseased, and this was confirmed by the subsequent necropsy, at which, however, the presence of a cerebral tumour was detected, namely, a psammona at the floor of the fourth ventricle, compressing the vagus nucleus. In cases of fæcal vomiting, supposed to be due to functional nervous disease, it would therefore always be advisable to examine thoroughly for signs of cerebral tumour (optic neuritis, &c.) and other organic nervous diseases.

In endeavouring to settle the functional nervous origin of a case of fæcal vomiting it may be very hard to exclude *simulation*, which, however unnatural it may seem, especially in the absence of any intelligible motive, has sometimes certainly been practised. Hysterical patients, like insane persons, are known to have swallowed fæcal masses before vomiting them, or to have placed fæces and enema material in their mouths and then pretended to vomit them. Mickulicz[2] recorded the case of a woman, aged 51 years, who had been operated upon five times before she was detected placing fæces in her mouth, and this detection saved her from a sixth operation. In a case of J. D. Bryant,[3] of New

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[1] *Société Médicale des Hôpitaux de Lyon*, October 13, 1908.

[2] *Deut. Med. Wochenschr.*, Vereins-Beilage, 1895, No. 18, p. 84.

[3] "Report of the Fourth Laparotomy on a Hysterical Patient," *Medical Record*, New York, December 24, 1902, p. 726.

York, four laparotomies were performed, the last because a communication between the stomach and colon was suspected. The patient, a young hysterical woman, aged 22, was afterwards discovered to have placed fæces and coloured enema material in her mouth in order to pretend to vomit them. In the discussion on Bryant's case Dr. A. H. Smith narrated the case of a woman with fæcal vomiting who had already had a laparotomy performed for the cure of a supposed gastro-colic fistula. A handkerchief full of fæcal matter was found concealed in her bed ready to be placed in her mouth or swallowed. Such cases belong to the class of *hysterical or neurotic simulation.* The patients seem often to have no intelligible motive for their deception, unless to become objects of interest or sympathy, and they seem rather to invite than fear the big surgical operations[1] that are likely to result from mistakes in diagnosis. They belong to the same class of patients as the young women sometimes detected in producing hideous eruptions or frightful ulcers by painful applications to their skins, apparently merely for the purpose of deceiving or of interesting their doctor and their friends.

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[1] A remarkable case of "hysterical malingering," narrated by A. A. Bowlby in the *Clinical Journal* (London, February 24, 1904, p. 292), illustrates the strange willingness of such patients to undergo operations. A young woman, about 30 years old, had previously had her left lower limb amputated near the hip-joint for supposed elephantiasis. She was under Mr. Willett's care at St. Bartholomew's Hospital for apparently a similar condition in the remaining (right) lower limb with which she said she had constant and extreme pain, and she was willing to have the limb amputated. The upper edge of the abnormal swelling was, however, very sharply defined, and one night the sister of the ward suddenly turned down the bed clothes and found a handkerchief twisted tightly round the limb like a tourniquet. The whole condition had evidently been artificially produced in this way by the patient herself. Latzko showed a woman at the Vienna Medical Club (abstract in *Muenchener Med. Woch.*, November 27, 1900), who, he thought, was feigning severe abdominal pain in order to have a laparotomy performed for the fourth time. It seemed that the first laparotomy had been performed for grave symptoms resembling those of gastric ulcer, but the operation showed nothing abnormal in the abdomen. The second laparotomy (of which no authentic account could be obtained) was declared by the patient herself to have been for nephrectomy. The third laparotomy was performed owing to ileus-like symptoms, and a left ovarian tumour was found and removed. Though the ovary was adherent to the sigmoid flexure no real obstruction to the passage of the contents of the gut was found.

Both "hysterical malingering" and functional nervous affections without malingering have given rise to striking instances of repeated surgical operations on the abdomen and elsewhere. Thoinot (*Société Médicale des Hôpitaux*, Paris, January 29, 1904), in a case of hysterical tympanites with right hysterical hemianæsthesia, explained the sequence of events as a result of suggestion. The patient it was known had undergone three laparotomies on the suspicion of tuberculous peritonitis. Thoinot supposed that the first operation relieved symptoms by suggestion, so that the patient at every relapse had energetically demanded another operation, and thus at the time was actually demanding a fourth operation, to which, however, Thoinot was not at all inclined to assent.

The difficulty in regard to the possibility of simulation is rendered still greater owing to the fact that persons with functional nervous symptoms sometimes afterwards simulate their old symptoms. This seems to have happened in Langmann's case already alluded to. In that patient, owing to the careful tests by coloured enemeta, &c., it can hardly be doubted that the fæcal vomiting was at first genuine, and, as the exploratory laparotomy showed nothing wrong with the abdominal viscera, the fæcal vomiting was apparently due to functional disease. Yet there was likewise simulation and hard fæces wrapped in paper were afterwards found under the patient's pillow. It seems indeed as if the wish to simulate all kinds of frightful diseases and court dangerous surgical operations depended on an abnormal condition of the nervous system similar to those neurotic states which lead to genuine hysterical vomiting, &c. It seems also as if the interest excited by their illness amongst those around them sometimes acts as an inducement to neurotic patients to simulate symptoms which originally were genuine, especially those symptoms on which the medical interest obviously centres. The incessant cross-questioning and suspicion to which such patients are often subjected may sometimes, by making them think of the whole matter, indirectly furnish them with the suggestion to simulate. In the case of Rosa S., which I have given at some length, there can hardly be a doubt that, although the fæcal vomiting was genuine, the patient tampered with thermometers, not only at the time of the fæcal vomiting, but also years afterwards when she was in the hospital under my care, and it is likewise possible that some of her "hysterical tympanites" was wilful.

I think it is quite clear that in the vomiting of fæces and enemata simulation can only be excluded by careful observation, that in some cases simulation has been detected, and that in others the precautions taken were not sufficient to exclude all possibility of deception. *Where, however, the requisite precautions can be taken, it is possible, by the use of coloured enemata, &c., to absolutely exclude all chances of simulation and render the diagnosis certain.*

I will conclude these remarks on diagnosis by adding that some cases supposed to be examples of *ordinary hysterical vomiting* ought really to be regarded as on the borderland between hysteria and malingering, and I will explain my reason for this belief. There certainly are some persons, especially young women, who, even whilst they are in their ordinary state of health and quite flourishing in appearance, have a really marvellous facility for vomiting.[1] Not only does the slightest temporary gastric upset cause them to throw up their food, but they have only to look at certain articles of food and think of the associated smells and tastes (for which they happen to have an aversion) in order to be able to vomit. This facility for vomiting may obviously be cultivated by mental processes, and may be temporarily greatly increased by causes which disturb the general health or the mental equilibrium. In fact, one must admit that some persons, at certain times at least, possess the power of voluntarily vomiting, that is to say, they can vomit merely as the result of a psychical condition, which they can themselves induce at will. It is therefore easy to see that great difficulties may arise when one has to deal with hysterical patients gifted with this power of vomiting, and at the same time desirous, as hysterical patients often are, of attracting attention to themselves by malingering if they cannot do so by other means.

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[1] It is of course well known that just as one race of animals differs from another by its facility for vomiting or the reverse, so in the human race some individuals vomit easily at the slightest cause and others only with difficulty. The cause which most readily produces vomiting in one individual is not necessarily the same as that which has most effect in another. In a conversation on the subject with my friend, Dr. H. Devine, he suggested that extreme facility for vomiting is comparable to the pow[illegible] of rumination occasionally possessed by human beings. I do not know [illegible]er the facility for vomiting is ever a family peculiarity, like rumi[illegible]ay be (*vide* "Bericht über eine Weiderkäuerfamilie," by Dr. [illegible]ller, in the *Muenchener Med. Wochenschrift*, August 5, 1902, [illegible] it would be interesting to find out whether it sometimes is so [illegible]

What portion of the Nervous System is chiefly at fault in cases of Fæcal Vomiting of Functional Nervous Origin?

S. Talma[1] observed that by irritation of the cœliac ganglion violent movements of a cramp-like nature in the intestines could be produced. The intestines sometimes remained contracted in many places, so that the lumen was quite obliterated. These cramp-like disturbances would pass off and then reappear again, and the direction of the movements was sometimes upwards and sometimes downwards.

In vomiting of functional nervous origin it is more probable that the main site of the disturbance in the nervous system is the brain. We know that violent vomiting is frequently a result of organic disease (tumours, &c.) and injuries (involving concussion) of the brain, and that vomiting of cerebral origin often occurs in the absence of any gross change in the brain (for instance, in simple concussion and in ordinary sea-sickness, and as a result of certain drugs, such as the anæsthetics chloroform and ether, which act on the cerebral cortex), and may even be due to purely psychical causes (such as ideas connected with disagreeable smells and sights, the sight of blood and surgical operations, and various mental emotions). Now, patients with functional nervous vomiting mostly exhibit characteristic signs of hysteria, and it is generally recognised, I believe, that the main symptoms of hysteria, such as hysterical hemianæsthesia, hysterical monoplegias and hysterical spasms, owing to their characteristic distributions, &c., are of cerebral cortical origin,[2] just as much so as the mental symptoms of hysteria are.

There is a good deal of special evidence pointing to the great influence of psychical states on digestive processes and movements of the alimentary canal. Everyone knows that mental conditions can have a great effect on appetite, and Professor J. P. Pawlow's[1] experiments on animals have shown the influence of the mere idea of food on the secretion of saliva and gastric juice. Parbon and Goldstein,[2] in their critical summary of the results of investigations regarding visceral representation in the brain, agree that not only are the gastric and salivary secretions influenced by cortical centres but that movements of the large intestine, rectum, &c., and other visceral functions, are to some extent represented in the cerebral hemispheres. Anger, fear, and other emotions have been proved to influence peristaltic movements in the alimentary canal of cats and dogs. W. B. Cannon,[3] whose investigations on antiperistalsis by the aid of bismuth food enemata and Röntgen rays have already been alluded to, observes that "there is no doubt that many emotional states are a strong stimulus to peristalsis, but it is equally true that other emotional states inhibit peristalsis." By his methods he was able to note that emotions such as anxiety, distress, or rage, caused a total cessation of the movements in the stomach and large and small intestines of the cats experimented on.[4]

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[1] *Deut. Arch. für klin. Med.*, Leipzig, 1892, vol. xlix., p. 206; quoted by Schloffer (*loc. cit.*).

[2] *Vide* S. J. Sharkey's summing up (BRAIN, Spring, 1904, p. 4) of the main reasons why th[illegible] brain, *i.e.*, cerebral cortex, is to be considered the part chiefly at fau[illegible] hysteria. Sharkey suggests that the reason why patients with hysteri[illegible]mianæsthesia are not usually conscious of their loss of sensation is t[illegible]ortical centres are the parts affected, whereas in hemianæsthesia [illegible]s brain disease the sensory fibres in the internal capsule are [illegible]ased and the cortical centres are sound, so that the patient pe[illegible]thesia.

If the almost physiological cerebral disturbances corresponding to ordinary fear or anger can thus influence the movements of the alimentary canal, how much more, I would ask, may the very pathological cerebral disturbances corresponding to the psychical states of hysteria be expected to do so? The intense functional disturbances of the brain occurring in hysteria are obviously never likely to be reproduced experimentally in animals. Experiments, however, as I have pointed out, do, to a certain extent, support the views expressed in regard to antiperistalsis of functional nervous origin.

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[1] "The Work of the Digestive Glands." English translation by Dr. W. H. Thompson, London, 1902.

[2] *Révue Neurologique*, Paris, 1903, vol. xi.

[3] *Loc. cit.*

[4] In regard to the influence of the brain on contraction of the muscular walls of the alimentary canal, the effects of drugs acting on the cerebral cortex, such as the anæsthetics chloroform and ether, may be borne in mind—not only the ordinary vomiting following the use of anæsthetics, but also the acute dilatation of the stomach (and possibly some of the grave cases of intestinal paralysis) occasionally noted after operations at which anæsthetics have been employed. W. Braun (*Free Meeting of Berlin Surgeons*, June 13, 1904) found that when he forced air through a previously established gastric fistula into the stomach of an anæsthetised dog great dilatation resulted, whereas the stomach of an animal not anæsthetised could not be dilated in this way because the air injected through the fistula was immediately expelled through the mouth by eructation.

I regard fæcal vomiting of functional nervous origin as simply a rare and extremely exaggerated form of ordinary hysterical vomiting, and I consider that hysterical vomiting is as much due to a functional disorder of the brain as the attacks of vomiting in a case of cerebral tumour are due to an organic disease of the brain. In regard to the violence of the symptoms in the functional cases it may be noted that in certain hysterical and neurasthenic individuals without organic disease the knee-jerk may be at times quite as exaggerated as in individuals with sclerosis of the lateral columns of the spinal cord. But why the vomiting in functional brain disease should be sometimes actually more violent and severe (fæcal vomiting) than it is in organic cerebral disease (fæcal vomiting practically never occurs in organic brain disease) I cannot understand, unless it be that disturbance of function can show itself in a more pronounced form in a brain without any gross organic disease than it can in a brain the finer mechanism of which has already been impaired by severe organic disease, such as cerebral tumour. In connexion with such a supposition one may remember that a delusion is apt to be less stable and not so well "organised" in a general paralytic, whose brain is the site of grave organic disease, than in a monomaniac, whose brain, could it be examined, would probably show no obvious organic changes.

Treatment and prognosis.—The best treatment is probably that by temporary isolation, together with the careful use of sedatives (bromides), lavage of the stomach, and by the mental and other treatment for hysteria in general, including fresh air, ordinary personal hygienic measures and the persistent avoidance of alcohol, tea and stimulating drinks, and of too stimulating and meaty foods. There is, of course, the usual danger of drug habits (especially morphinomania) becoming developed.

It has been said that the subjects of the gravest forms of hysteria sometimes end by becoming inmates of an asylum. Doubtless they may sometimes become inmates of asylums like the man and the woman whose cases Sander (*loc. cit.*) described.[1] The patient, Rosa S., however, when I saw her six years after her laparotomies, though again suffering from some of her old troubles, had apparently during the interval been able to earn her own living and give satisfaction in domestic service.

MAIN CONCLUSIONS.

(1) Functional nervous vomiting, like the hemianæsthesia, palsies and spasms of hysteria, must be regarded as due to an abnormal state of the cerebral cortex, and is just as much a symptom of functional brain disease as the vomiting in cases of cerebral tumour is of organic brain disease.

(2) Fæcal vomiting of functional nervous origin is merely a rare and extremely exaggerated form of ordinary hysterical vomiting.

(3) The vomiting in functional brain disease may sometimes be more violent and severe than it ever is in organic cerebral disease, since fæcal vomiting is scarcely known to occur in cases of cerebral tumour, &c. Some light is thrown on this point by the fact that a delusion is apt to be more stable and better "organised" in a monomaniac whose brain, could it be examined, would probably show no obvious change, than in a general paralytic whose brain is the site of grave organic disease.

(4) For the occurrence of fæcal vomiting of functional nervous origin active intestinal antiperistalsis is absolutely necessary. But it is not certain that antiperistalsis necessarily always plays a part in the fæcal vomiting known to surgeons as a symptom of organic intestinal obstruction (organic ileus).

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[1] "Hysterical Malingerers" are, of course, not unlikely at some period of their career to get shut up in an asylum, or even in a prison or penitentiary. *Cf.* the discussion on the case recorded by Dr. J. D. Bryant, of New York (*loc. cit.*). Malingerers, hysterical or not hysterical, who from quite intelligible motives are willing to undergo grave surgical operations (though they do not court them), are, naturally, occasionally to be found amongst both sexes in prisons, and even amongst soldiers in countries where conscription exists.

(5) The fæcal vomit in organic obstruction of the bowel is seldom, if ever, more than "fæculent," that is to say, having the odour of fæces without containing obvious (visible) fæcal particles or masses. Vomiting of formed fæces, in the absence of malingering and gastro-colic fistula, practically only occurs in functional nervous cases. This may partly be accounted for by remembering that antiperistalsis, if it occurs at all, is likely to be more forcible when the muscular walls of the gut have not been previously weakened by overdistension or gross organic disease.

(6) "Hysterical Malingering" is of course apt to develop in the same (hysterical) class of patients in whom fæcal vomiting occurs, and the possibility of genuine fæcal vomiting occurring side by side with simulation must be kept in mind.

INTRAMEDULLARY ABSCESS OF THE SPINAL CORD. AN ACCOUNT OF THREE CASES.

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AND

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Suppuration within the spinal cord is a rare event, except when occurring in the course of suppurative leptomeningitis. During the past two years three cases have come under our observation, which seem to us worthy of being placed on record, not only on account of the rarity of the pathological condition, but also since the location of the intramedullary abscess and its path of extension show certain features in common with those of syringomyelia and hæmatomyelia. Further, the method of extension of the intramedullary abscess presents certain points of interest. We are indebted to Dr. Beevor for his kind permission to make use of the notes of two of the cases, to Dr. Ferrier for the third case, and to Dr. Farquhar Buzzard, who made the pathological examination upon one of the cases.

We have made a careful search through neurological literature, but we have found no description of cases of a nature similar to those here recorded. Sir William Gowers describes intraspinal abscess as occurring only in the course of suppurative meningitis, and this appears to be the opinion generally held.

The three cases here described resembled one another closely. They were cases of pressure or evascularisation paraplegia, in which a transverse portion of the spinal cord was the seat of myelitis or ischæmic necrosis. The local necrotic lesion resulted in the first case from the pressure of a new growth of carcinomatous nature, in the second case from tuberculous pachymeningitis, associated with spinal caries, and in the third case from the pressure of extrathecal tuberculous matter, associated with old spinal caries.

In all these cases suppuration, commencing doubtless in a necrotic region of limited extent, spread in the length of the spinal cord far above and below the area of original damage, its distribution being strictly limited and unassociated with any "myelitic" change.

In the *first* case the paraplegia was slow in onset and had been complete for several weeks when suppuration occurred. Though it is certain from the pathological examination that the suppuration occurred subsequent to the evascularisation, yet nothing in the clinical history of the case seemed to indicate at what period suppuration commenced. The physical condition was feeble. Rigors did not occur, nor was the temperature above normal throughout. There was no alteration in the spinal symptoms indicative of the upward extension of the abscess.

In the *second* case the paraplegia became complete in a few days after the first appearance of spinal symptoms, and this was evidently the result of extensive strangling of the spinal arteries by pachymeningitis in the lower half of the dorsal region. The patient lived a month after the onset of paraplegia. The advent of suppuration was presumably marked by the occurrence of pyrexia and rigors, which persisted till death, but by no other change in the clinical picture, referable to the nervous system.

In the *third* case the onset of paraplegia was rapid. The presence of pyrexia during the time he was under observation, and the fact that the spinal symptoms progressed as if from a spreading lesion, made it probable that suppuration rapidly followed the occurrence of myelitis and that the spread of symptoms was evidence of its extension. That suppuration commenced subsequently to the occurrence of myelitis is, we think, clearly proved by the fact that in the limited transverse area of the compression-myelitis the spinal cord was completely necrotic, and at this point the abscess was least in evidence, tissue reaction being impossible in the necrotic area; but above and below the lesion, where the abscess was conspicuous and extended for many inches in the length of the cord, there was no sign of such necrosis.

It is remarkable that in the first two cases no extension of the spinal cord symptoms was noticed correlative to the upward extending abscess. It seems to us probable in these cases that the abscess extended rapidly in a few days before death, when the psychical and physical condition of the patients debarred examination. Moreover, the extension of an intramedullary lesion is apt to be unaccompanied by any subjective phenomena such as would appeal to the consciousness of a moribund patient. We think also that the rapidity of pus formation in the spinal cord is apt to be festinant, since there is little resistance to the spread of fluid at a pressure along certain tissue lines in the spinal cord. Therefore the larger the pyogenic membrane the more pus formed, the higher the potential pressure and the more rapid the extension of the pus along the clearage lines. This supposition is rendered the more probable since we found the abscess wall ill-marked, or absent, at the upper and lower limits of distribution.

The abscess formation in these three cases showed several points of interest. In the region of necrosis the pus was not limited to a definite abscess, but occupied certain interstices of the sponge-like necrotic tissue, the largest collections in the necrotic regions being in the position of the lateral pyramidal tracts. This fact is interesting since the lateral regions of the spinal cord are invariably the earliest regions of the spinal segments to show degeneration as the result of pressure and evascularisation, and it is possible that infection occurred in the region of earliest degeneration. There were no signs of inflammatory reaction round the pus collections in the necrotic areas, but the pus occupied as it were the interstices of a sponge. Immediately above and below the necrotic areas, the pus was confined by a well-marked abscess wall round which were marked reactionary changes, the vessels being engorged and some of them occluded, while there was a considerable aggregation of small round cells. As the abscess in each case was traced towards its upper and lower limits, the wall became less marked and disappeared near the extremities, a small-celled infiltration of the white matter marking the poles. It seemed certain that though the abscesses had chiefly extended by cleavage of the tissues, yet there was some definite destruction of the white matter in the formation of the abscess. The fact that the contents of the abscess, especially in its more recent portions, showed numerous points which stained deeply by Weigert's method, also argues that disintegration of the white matter occurred in the process of formation of the abscess.

In certain places the abscess wall seemed to have disappeared, where there was evidence that a process of degeneration was extending from the abscess into the posterior columns. Otherwise the very slight amount of change noticeable in the grey and white matter of the spinal cord was remarkable, the abscess having displaced and compressed rather than destroyed these tissues.

Cultures from the contents of the abscess were not made in any of the cases.

Sections of formalin-hardened material stained by Loeffler's method showed that both staphylococci and streptococci were present.

Distribution.—The abscess cavity was in each case (except in the necrotic areas), confined to the posterior columns and posterior horns. At the upper limit it was mesial in the centre of the posterior columns. In the lower cervical area a tendency is apparent for the abscess cavity to split into two, the division being referable to the greater resistance of the posterior median septum in this region. In Case 1 the abscess cavity was double in the cervical region and confined to the posterior horn of either side. The position of the abscess in the dorsal region was the same in all the cases, viz.: at the junction of the posterior horns and posterior commissure, involving both the grey matter of the posterior horn and the white substance of the posterior column.

In Case 1, where the abscess extended into the lumbosacral region, it occupied almost the whole area of the posterior columns.

The tendency of syringomyelic cavities and intramedullary extravasations of blood to occupy positions close to the dorsal septum and posterior horns, as did the abscess cavities in these cases, is probably owing to the fact that there is less resistance to cleavage and less tissue resistance in these situations, since these are the regions occupied at one period of fœtal life by the lateral and posterior limbs of the medullary canal, which subsequently become obliterated. Moreover, it is held that the periependymal neuroglia may persist after the obliteration of the limbs of the medullary canal, and may by subsequent development give rise to syringomyelic cavities.

The long posterior limb of the fœtal medullary canal is transformed into the posterior median septum. The anterior shorter limb persists as the medullary canal, while the lateral limbs which stretch along the posterior horns for some distance close to the posterior columns become obliterated.

We submit, since cystitis was present in each of our cases, that the bladder may have been the place of entrance of the organisms of suppuration into the system, and that their incidence upon the spinal cord was determined by the lowered vitality of certain regions of the spinal cord by pressure and evascularisation, and that the point of origin of the abscesses was in the necrotic region.

Case 1.—M. C., aged 61 years, was admitted into the National Hospital, under the care of Dr. Beevor, on August 10, 1902, suffering with paraplegia. The illness began in September, 1901, with severe pain in the shoulders, running down the right arm. At Christmas, 1901, weakness of the legs appeared. In April, 1902, atrophic palsy of the hands was noticeable and she became unable to stand.

When admitted there was considerable general wasting. There was complete loss of sensibility to all forms of stimulation below the level of the second costal cartilage and over the

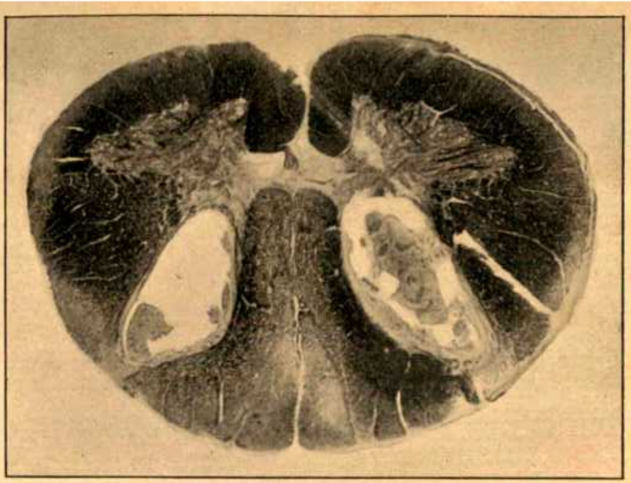


FIG. 1.

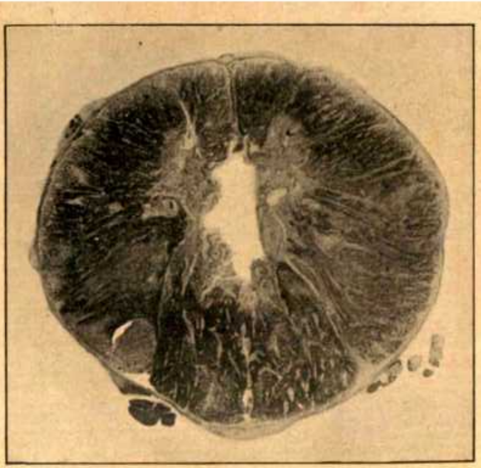


FIG. 2.

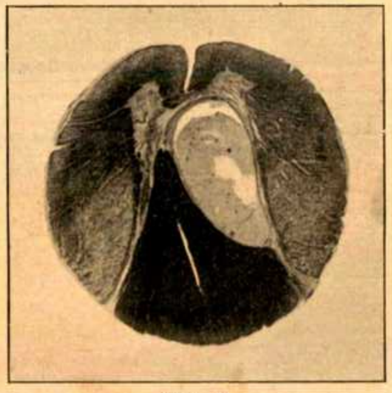


FIG. 3.

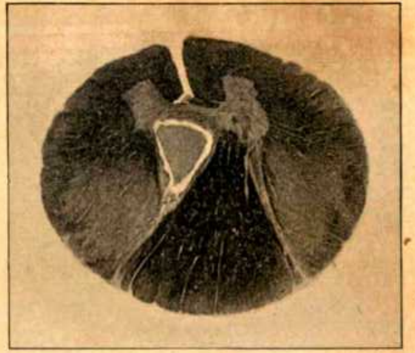
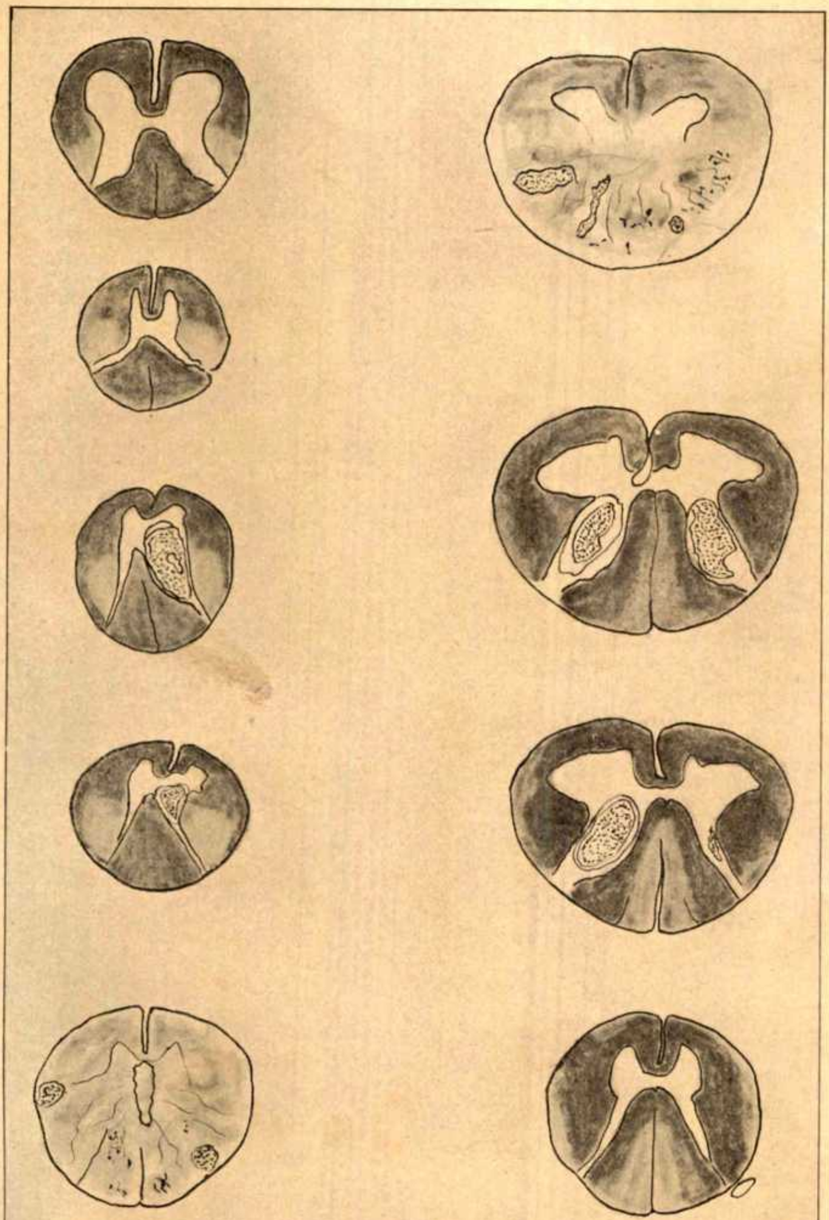


FIG. 4.

Photographs of spinal cord of Case 1.

PLATE ~~IV~~.II.



Projection drawings from Weigert-Pal specimens of Case 1.

Sections stained by Loeffler's method and by carbol-thionin revealed the presence of staphylococci and streptococci.

Case 2.—M. P., aged 37 years, was admitted into the National Hospital under the care of Dr. Beevor on August 22, 1902.

Her illness began in September, 1901, with pain in the lower part of the spine. The pain persisted and was not increased by movement of the spine. A month before admission on attempting to get out of bed one morning she found that she was unable to move the legs, and had lost the feeling below her waist and had incontinence. This was the first sign of paralysis noticed. After this she became gradually weaker, the paralysis remaining absolute. She suffered a good deal from delirium. When admitted there was much general emaciation, the lower extremities being especially wasted. Her mental condition was stuporous. There was absolute loss of sensibility below the third costal cartilage, and complete atrophic paralysis of the lower extremities with loss of all the reflexes. There was no sphincter tone.

There was some deformity and tenderness of the ninth dorsal spine.

Fourteen days after admission the temperature rose suddenly to 101° F., and continued irregular and intermittent, reaching on occasions 105° F. On two occasions rigor occurred. She died on August 30.

Autopsy.—Examination of the vertebral column showed that the disc between the ninth and tenth dorsal vertebræ had been destroyed by tuberculous disease. The bodies of the vertebræ were much diseased and a sharp projecting ridge formed by their contiguous edges pressed upon the cord. The theca was affected with tuberculous pachymeningitis, and was adherent to the bony canal from the seventh dorsal to the first lumbar vertebræ.

The spinal cord was of normal size and appearance in the cervical region only. Below the level of D. 6 it tapered rapidly. In the lower dorsal region it was shrunken and brown in colour, and was evidently completely necrotic. The same change was noticeable, but to a less extent, in the lumbo-sacral region. The nerve roots of these regions were much matted and many of the vessels were thrombosed. (Plates III. and IV.)

The first dorsal segment was soft and fluctuating to the touch, and a section across the cord at this level revealed an abscess, circular in transection and of diameter 4·5 mm. (Plate IV.)

Further section of the cord showed that the abscess, which contained creamy-white pus, extended from the lower part of the third cervical segment to the conus medullaris. Its limits are shown in plates III. and IV. From the sixth dorsal segment to the first lumbar segment the cord was completely necrotic from evascularisation myelitis. At its upper limit and in the necrotic region the abscess showed no marked wall, but in the lower cervical and upper dorsal region there was an obvious wall.

In some places, however, the wall was absent, and here the infection necrosis was evidently spreading directly into the posterior columns.

Marked ascending and descending degeneration in Weigert-Pal specimens showed that the ischæmic necrosis had preceded the abscess formation by some considerable time.

Case 3.—R. C., aged 17, was admitted into King's College Hospital, under the care of Dr. Ferrier, on December 29, 1903, suffering from loss of power in the legs and retention of urine of three days' duration.

About nine years ago the patient had suffered from curvature of the spine, the result of which was visible in a large antero-posterior curve, whose most prominent point was formed by the spinous process of the fifth dorsal vertebra. There had been no loss of power in the legs previous to the present attack, which came on suddenly during the night of December 26, 1903.

December 29, 1903. On admission, it was found that the patient had lost all voluntary power of the lower limbs, which were in a state of flaccid paralysis. The knee jerks were not obtained, there was no ankle clonus, and both plantar reflexes were of the extensor type. There was complete anæsthesia to all forms of sensation as far up as the level of D. 8. There was complete retention of urine, requiring the use of the catheter. The urine was normal.

January 1, 1904. A further examination on this date showed that the upper limit of anæsthesia had ascended to the level of the fifth rib.

January 4. The anæsthesia was noted as having extended to the upper border of the second rib. There was also some impairment of the fine movements of both hands, more especially of the fingers and thumb. In addition, there was a well-marked band of anæsthesia along the ulnar aspect of both arms, forearms and hands. Some cerebro-spinal fluid, withdrawn on this date by lumbar puncture, showed about 1 per cent. of albumen, a few leucocytes and no bacteria. Cultures remained sterile for two days. The patient remained in this condition for the remainder of the month of January. During this period the temperature was of a hectic character, rising from normal in the morning to 102° or 103° F. at night.

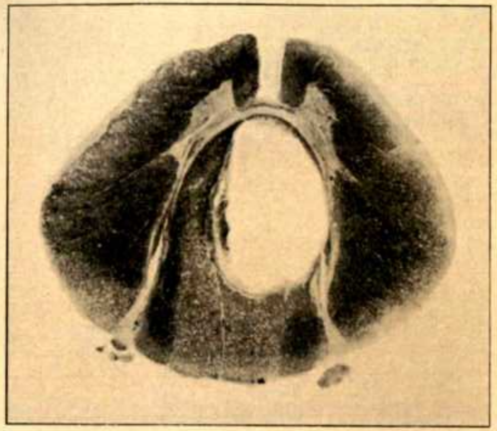


Fig. 1.

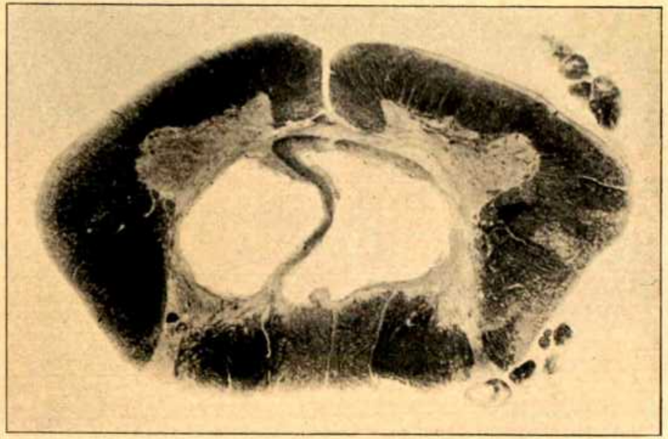


Fig. 2.

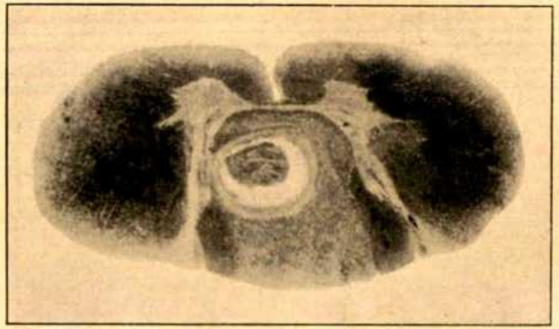


Fig. 3.

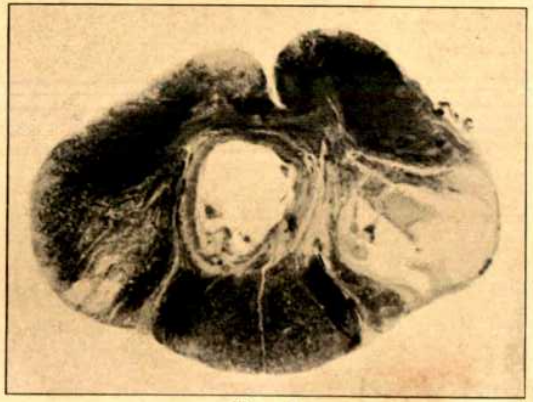
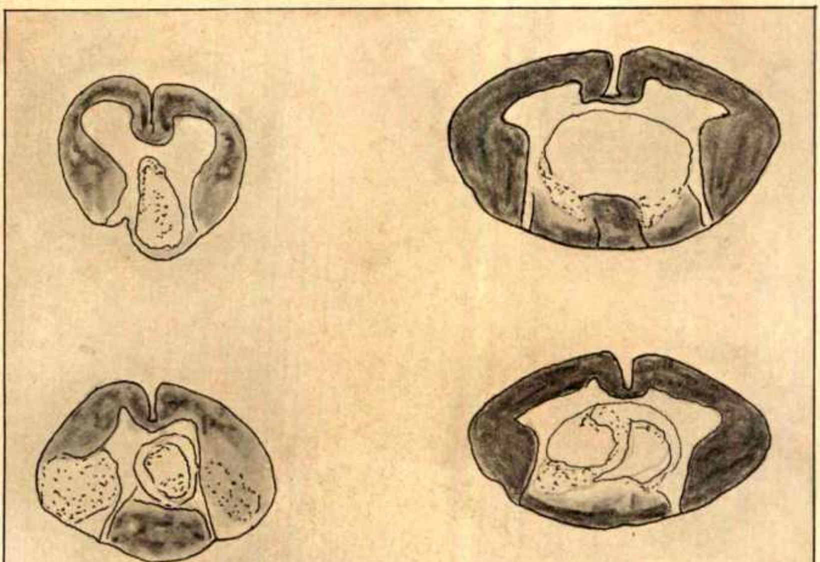


Fig. 4.

Photographs of spinal cord of Case 2.

PLATE ~~II~~.IV.



Projection drawings from Weigert-Pal sections of Case 2.

February 24. By this date the anæsthesia had disappeared from the arms and the grasps had improved in strength. The upper limit of anæsthesia upon the trunk remained at the second rib. The temperature continued to be of the intermittent type.

There was practically no change until March 16, when signs of pneumonia and œdema of the pulmonary bases ensued, and he died on March 18, 1904.

The *post-mortem* examination showed an extensive angular curvature of the dorsal portion of the spinal column with much cario-necrosis of the bodies of the sixth to the ninth vertebræ. The cord was completely disorganised, and at the level of greatest curvature was much reduced in size and surrounded by thickened dura mater.

The lungs showed adhesions at the apices, with a recent purulent pneumonia at the right base and œdema, and collapse at the left.

Microscopic Examination. (Plates V. and VI.)

After hardening in formalin, sections of each segment of the spinal cord were prepared by the hæmatoxylin-eosin method.

There was complete transverse necrosis of the eighth dorsal segment sharply limited below, while some patches of softening were apparent in the seventh and sixth dorsal segments, the highest seat of softening being the left lateral column of the fifth dorsal segment. Below the eighth dorsal segment the spinal cord presented no peculiarity. The necrosis in the eighth dorsal segment was apparently an evascularisation necrosis, and in this segment no signs of pus-formation were present.

In the seventh dorsal segment pus was present in the interstices of the softened areas at the points of entry of the left posterior and right anterior roots, and Dr. Buzzard considered that the path of infection was presumably by the vessels accompanying these roots.

In the sixth and fifth dorsal segments the collection of pus, which has been described as occupying the region of the entering left posterior root in the seventh dorsal segment, crept gradually along the limit between the left dorsal horn and left posterior column to gain the region of the posterior commissure where, in the fourth dorsal segment, it assumed a rounded form about 1·5 mm. in transverse diameter and presented a definite abscess wall. (Plate VI.) In the second dorsal segment the abscess was of much greater size and extended from point of entrance of the right dorsal root to the left cornu-commissural junction, involving

PLATE V.

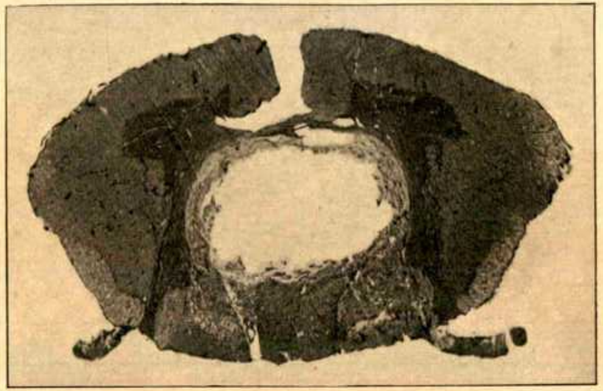


Fig. 1.

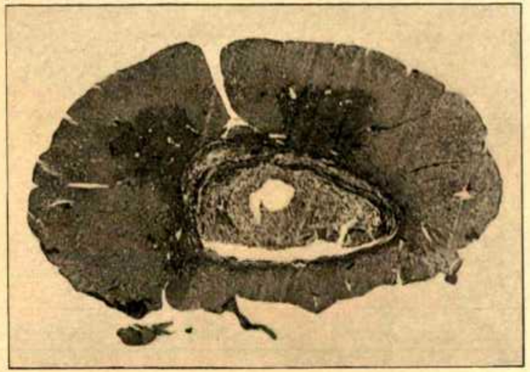


Fig. 2.

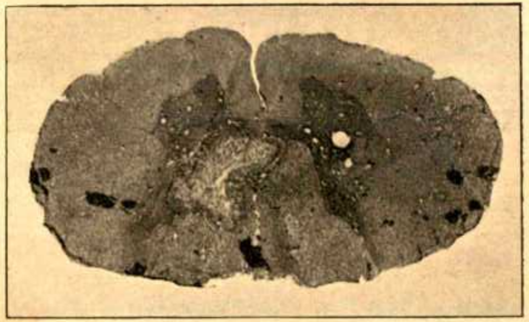


Fig. 3.

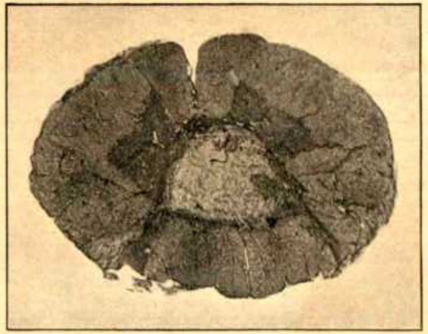
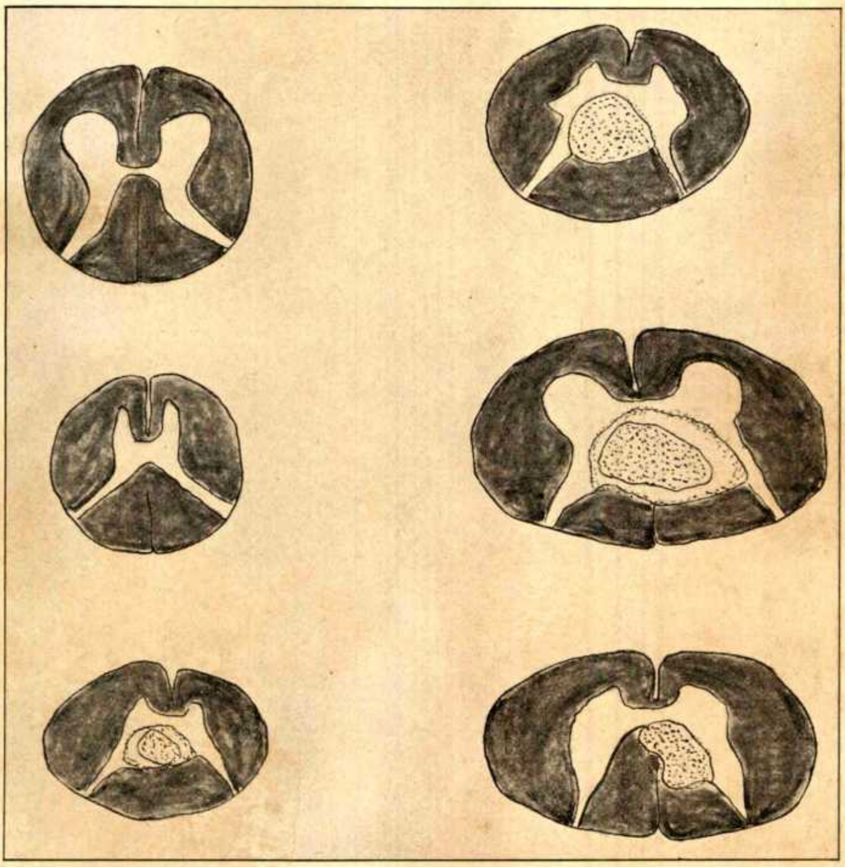


Fig. 4.

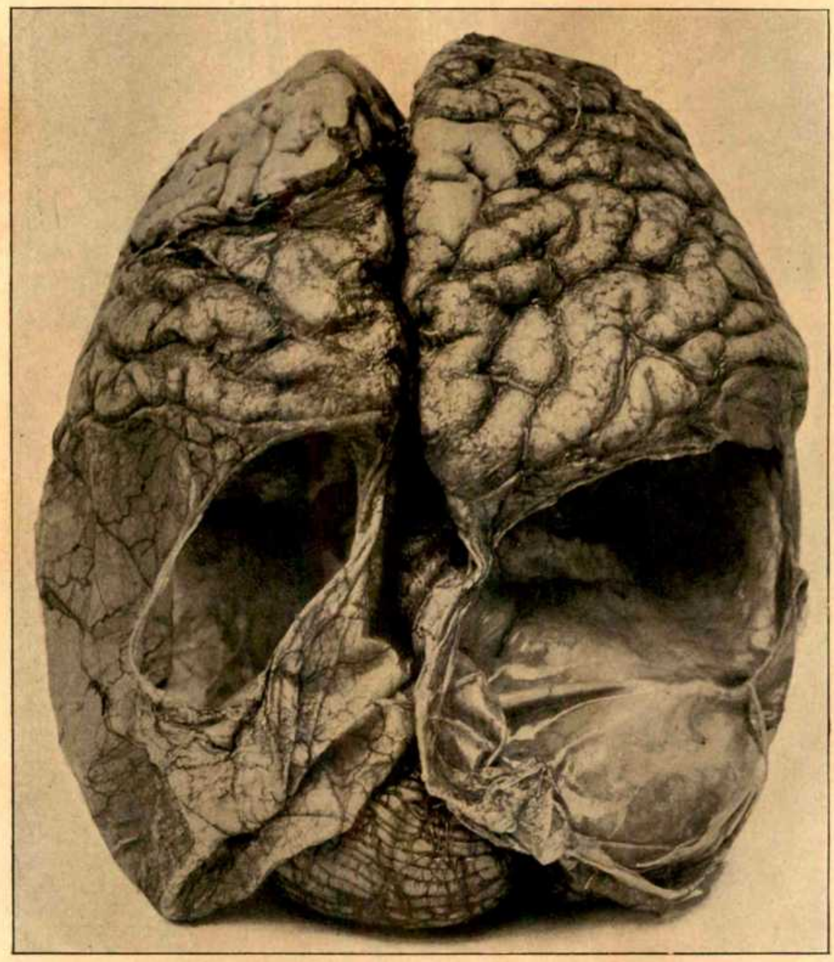
Photographs of spinal cord of Case 3.

PLATE VI.



Projection drawings from hæmatoxylin eosin specimens of Case 3.

PLATE I.



View of brain from above.

(From a photograph taken immediately after removal.)

BILATERAL LOSS OF POSTCENTRAL CORTEX, APPARENTLY CONGENITAL, IN AN ADULT.

BY HUBERT M. TURNBULL, M.A., M.B. (OXON.).

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II.—Life history.

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III.—Macroscopical examination of (1) cysts, and (2) cortex.
IV.—Microscopical examination of (1) cysts, and (2) cortex.
V.—Macro- and micro-scopical examination of parts of the central nervous system, other than the cysts and cortex.

(*C*) REMARKS.

VI.—Changes secondary to the primary cortical lesion.
VII.—The primary cortical lesion.
VIII.—Physiology.

(*A*) INTRODUCTION.

THE patient, Annie Lawrence, aged 24, was admitted, suffering from severe burns, to the London Hospital, under the care of Mr. Mansell Moullin, January 17, 1903. She was greatly collapsed and died within a few hours. A *post-mortem* examination was made on January 19.

I.—EXTRACT OF THE NOTES MADE AT THE NECROPSY.

External appearances.—The entire body and face were covered with burns of the second and third degree.

The breasts, external genitals and pubic hair were well developed.

The arms were flexed at the elbow beyond a right angle, the wrists strongly flexed and adducted, the fingers flexed.

The legs showed no marked wasting, but the feet were rigidly fixed in the position of talipes equinovarus.

Skull and brain.—The skull cap presented no abnormality superficially, but the whole skull was smaller than normal, and on section to remove the calvarium no diploe was seen in the uniformly hard bone.

On opening the skull, over half a pint of perfectly clear fluid escaped.

On removing the dura, a large cyst was seen on each side completely occupying the space for the occipital lobe; one of these cysts had been torn in removing the skull, allowing its contents to escape.

The pia over the remainder of the brain peeled very readily, was very much engorged and somewhat opaque.

The anterior clinoid processes of the sella turcica were much turned back. The pituitary body was small but apparently normal.

The brain, which weighed 21½ ozs., was photographed after removal (plates I. and II.), and preserved in 5 per cent. formalin. The spinal cord was placed in Müller's fluid.

Thorax and abdomen.—On removing the sternum a mass of thymus 1½ ins. long by 1 in. wide was seen. The lower lobes of the lungs were congested; the other thoracic and abdominal organs were healthy.

Pelvis.—The sacral promontory was unusually prominent, the true conjugate measuring 3⅞ ins. Both ovaries were large and very soft.

II.—The Life History.

The life history was obtained from the mother (February 25, 1903), the elder sister (April 8, 1903), and Mrs. H. (March 4, 1903), the landlady in whose house the patient had been since birth. To ensure accuracy each person was interviewed alone. It has been impossible to trace the doctor who attended the patient most of her life.

The family history.—The father died seventeen years ago, but never had any mental symptoms. The mother, aged 47, is healthy and has had no miscarriages, but three children :—

Charlotte (born 1876), healthy and intelligent;
Annie (born 1878), the subject of this paper;
A boy (born 1880), who died as an infant of bronchitis.

History of birth.—The patient was delivered, without instrumental help, in an easy labour. A few weeks later the landlady, noticing that the baby did not follow people about with her eyes, suspected blindness, and the baby was taken to Moorfields, where the surgeon said that she was quite blind and that nothing could be done for her sight. The out-patient notes at Moorfields are destroyed after ten years, so that no accurate report of the examination can be now obtained.

Fits.—As a baby she had several infantile convulsions, which yielded readily to immersion in a warm bath.

Later she had two severe fits with unconsciousness, which the doctor said were epileptic; the second occurred before the death of the father seventeen years ago, but the exact dates are not known.

She had numerous fits of temper excited either by no apparent cause or by her being thwarted in some way. In these attacks she would cry, scream, and strike at people, attempts to pacify her only augmenting her excitement. Her tongue was never bitten, urine was never passed, and when the fit ceased she did not sleep, nor did she appear in any way unusual.

She never had attacks of paralysis or transitory weakness, but her sister thinks she became gradually weaker latterly.

Vomiting.—She vomited a good many times, but apparently from stomachic causes.

Vision.—She never showed signs of seeing anything, never examined anything with her eyes, never watched people about the room nor pointed at anything. She simply stared straight ahead, but her eyes were "exactly like an ordinary person's," and there was no squint.

Speech.—She was late as a child in uttering recognisable words, but the range of her speech increased gradually and progressively; there had never been any loss, sudden or gradual, of powers of speech.

She spoke intelligible words, for not only members of her family but strangers had no difficulty in understanding her.

She used to talk for hours to herself, repeating whole sentences strung together without reason. She would start speaking when others talked, and more especially whispered, in the same room; if, however, the conversation was on general topics, as a rule she took no notice, but the mention of food always excited her to join in, repeating what she heard.

She could answer some questions sensibly and correctly in the affirmative or negative, as for instance, if her sister asked, "Has mother gone to fetch her work home?" or, "Does Annie like fish?"

In reference to her food she is said to have got beyond mere answers in the negative and affirmative, thus she would say when fish was being fried, "Annie is going to have some fish," and if she had pudding she would repeat, "Annie doesn't like mother's pudding, Annie likes Mrs. H.'s pudding." Apparently these were repetitions of words addressed to her at the time, which ultimately became associated in her memory with the respective dishes.

Words, too, apparently became associated with acts; if, for instance, her sister said "Lotty is going to bring Annie home so and so," she not only repeated the words, but if her sister returned subsequently without the promised dainty, she would have a fit of temper.

She never addressed her mother or sister by name, never expressed her bodily feelings, saying she was hot, cold, hungry, in pain, desirous of micturating, &c.; she never made any demand and no verbal order was ever given to her. She never talked of the actions of herself or others.

No attempts had been made to educate her at home, and the School Board for London state that the patient had never attended either of the schools for the blind, as she was not only blind but imbecile from birth.

Hearing.—Her hearing was acute.

Not only could she hear, but she understood the significance of sounds. Thus she appreciated the sounds caused by the preparation of food and always had a fit of temper when she heard the floor being washed. She recognised the approach of strangers even when still outside the room, and would shout and scream. The approach of some occasional visitors—for instance, the doctor—she did not resent.

Tactile sense.—She never felt the faces, &c., of different members of the family, and touch played no part in their recognition. Some foods when placed in her hand she never attempted to eat, others she ate greedily, but she was probably guided by her sense of smell. She never attempted to eat her toys. She was constantly playing with objects, swinging them about aimlessly and changing them from hand to hand, but though she had several toys she gave no evidence of recognising or preferring any of them—a door key or old nail being as welcome as her own dolls.

Sense of smell.—She used to smell most of her food before eating it, and she had definite tastes in food, hardly ever eating her bread and butter unless a piece of meat was added. Fish she was very fond of, fruit she never ate.

Taste.—She would spit out food she did not like and never swallowed anything sweet.

Expressions.—She would smile when pleased, but never laughed heartily. If upset she would weep and scream and hit out. Her tongue was not lolled out.

Sphincters.—In process of time she learned to defæcate and micturate when placed on the stool, and never soiled herself unless she had diarrhœa. She never, however, made any signs of desire to relieve herself.

Motion and Gait.

Legs.—As a child she used to walk about by herself in the garden and walked with her mother and sister every Sunday to an aunt. Coming home one Sunday, however, twelve years ago, she had an ordinary fit of temper and had to be practically carried home. Both the mother and sister are certain that her refusal to walk was not due to sudden weakness or paralysis, for she not only screamed but struggled violently with both arms and legs as they dragged her home. This was at least five years after her second epileptic fit. After that day she became violent if any attempt was made to get her to go for a walk, and her mother soon left her to sit all day undisturbed. Latterly she never walked spontaneously, but remained sitting on the floor wherever placed, and to move her from one spot in the room to another an arm was placed under her shoulders; thus supported she stepped out correctly, but her legs seemed very weak. Her burns were apparently due to the fall of a cinder from the fire, close to which she had been left sitting.

There were no movements of the legs when she was sitting.

Arms.—She could neither dress herself, do her hair, button her clothes, nor blow her nose. She could eat food placed in her hands, but could not manipulate a cup, knife or fork. She was constantly lifting small objects and toying with them about her face. She often scratched her head and would strike out when in a temper, although not destructive in her habits. The muscles of her arms were always on the move except when she was asleep. These movements were not repetitive, nor rhythmical, but she "seemed always to be testing her muscles."

Her sister thinks that she got weaker latterly.

Contractures.—She sat usually tailor fashion, and once had a sore on an external malleolus. Though her sister constantly washed her hands she never noticed any abnormality in them, save that they were small and the skin much more delicate than her own.

Sensations.—She never complained of pain, headache, heat nor cold.

Memory.—In addition to instances of memory already mentioned, she always showed signs of expectation on Sunday for a pudding presented weekly by the landlady.

Menstruation.—She menstruated regularly.

Illnesses.—She was treated for bronchitis as an out-patient at the Great Eastern Hospital for Children, but the notes have been since destroyed.

(*B*) MORBID ANATOMY.

MACROSCOPICAL EXAMINATION OF (1) THE CYSTS, AND (2) CORTEX.

(1) *The Cysts.*

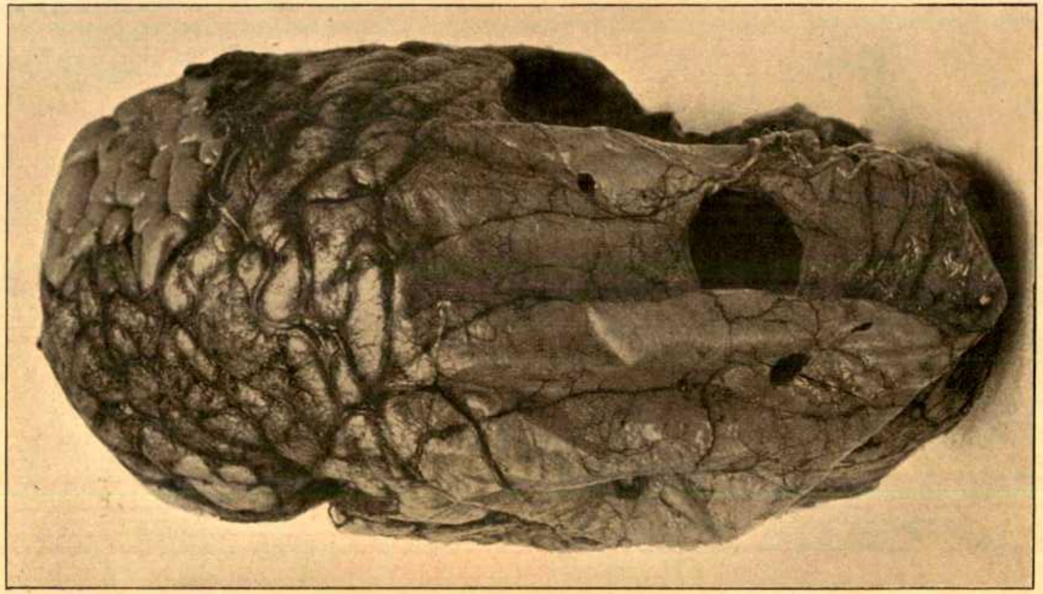
The cysts, seen in the *post-mortem* examination, formed the walls of the posterior portion of the right and left ventricles, and it was apparent that the cysts represented enormously attenuated portions of cortex, each cyst being covered by pia-arachnoid and presenting a smooth glistening surface towards the ventricular cavity (*vide* frontispiece, plate I. and plate II.).

When the pia-arachnoid was stripped off, the cyst wall was seen to be a very thin transparent membrane with smooth and glistening surfaces of a colour similar to the cortex.

On the right side the Limits of the Cyst and its Mode of Origin from the thick Cerebral Cortex were as follows:—

On the outer surface above the level of the fissure of Sylvius the cyst seemed to spring from the summit of the cortical convolutions lying immediately anterior to it (*vide* plate II.), but dissection showed that it sprang from the bottom of a deep fissure and passed directly upwards, separated from the cortex anterior to it by a layer of pia-

PLATE II.



External view, cyst of left hemisphere.

(*From a photograph.*)

mater, until reaching the level of the exposed surface of the brain it turned sharply backwards. This fissure was formed by the junction of the upward turn of the posterior limb of the Sylvian fissure (*s.*, fig. 1) with the inferior and superior postcentral moieties of the intraparietal fissure of Turner (*s.pc.s.*).

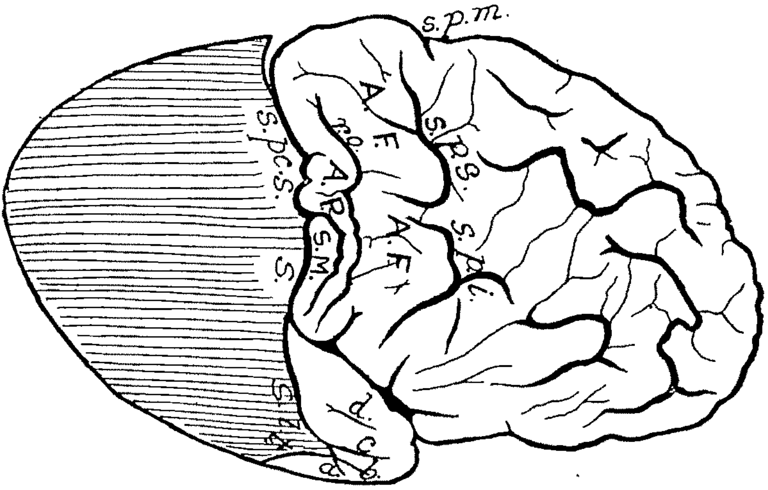


FIG. 1.

External aspect of Right Hemisphere (reduced from a drawing. Cyst wall shaded).

A.F., ascending frontal convolution. *A.P.*, ascending parietal convolution. *S.M.*, anterior limb of supramarginal convolution.

s.p.m., sulcus præcentralis medialis. *s.p.s.*, superior præcentral fissure. *s.p.i.*, inferior præcentral fissure. *r.o.*, fissure of Rolando. *s.*, the upturned end of the fissure of Sylvius joining *s.pc.s.* the superior postcentral fissure and upper part of the inferior postcentral fissure, which continues between *A.P. and S.M.* *s.l.t.*, fissure separating remnant of temporal lobe from cyst.

a. b. c. and *d.* mark the four shallow fissures on the remnant of the temporal lobe.

For one centimetre behind its point of origin the cyst wall was thickened sufficiently to cause a slight opacity.

At and below the level of the fissure of Sylvius the cyst had origin from the postero-superior angle of the intact isle of Reil, and from a shallow but definite fissure limiting the remnants of the temporal lobe posteriorly (*s.l.t.*).

This fissure is seen (figs. 1 and 5, *s.l.t.*) to turn back at the temporal pole enclosing an angle; the cyst wall in this angle was opaque owing to the presence on its outer surface of a thin layer of cortical substance 2 cm. in extent. Moreover, the remnant of temporal cortex thinned out rapidly posteriorly, so that here the transition from cortex to cyst was more gradual.

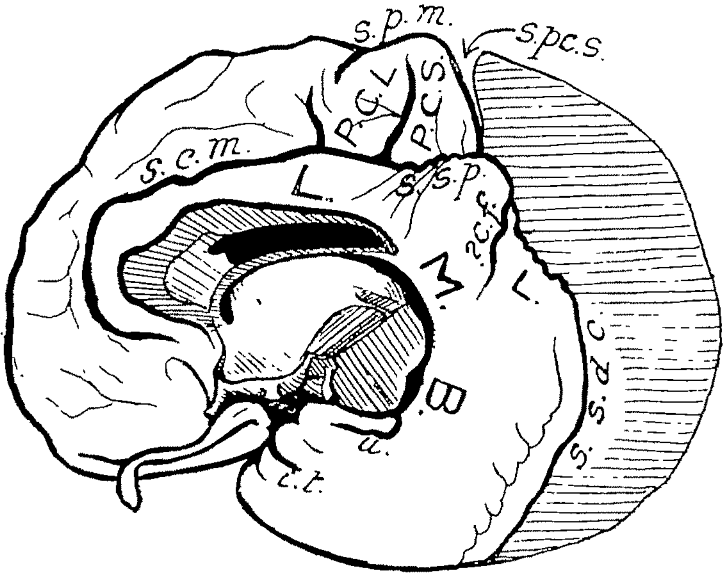


FIG. 2.

Inner aspect of Right Hemisphere after removal of hind brain (reduced from a drawing. Cyst wall shaded).

P.C.L., paracentral lobule. *P.C.S.*, part of the precuneus. *L?* portion of limbic lobule. *L. M. B.*, limbic lobe.

s.p.m., sulcus præcentralis medialis. *s.c.m.*, callosomarginal fissure, the upturned end of which passes between *P.C.L. and P.C.S.* *s.s.p.*, subparietal fissure. *? c.f.*, probably the anterior limb of the calcarine fissure. *i.t.*, incisura temporalis. *u.*, uncus. *s.s.d.c.*, shallow fissure defining cyst from thinned-out cortex anterior to it.

On the inner aspect of the hemisphere (fig. 2) the superior postcentral fissure (*s.pc.s.*) cut down to the subparietal fissure (*s.s.p.*) sharply separating a small portion of precuneal lobule from the cyst. Below the level of the subparietal fissure, which was in direct continuity with the callosomarginal fissure (*s.c.m.*), the cortex swept round the splenium of the corpus callosum as a continuous band, corresponding to the posterior portion of the limbic lobe, to terminate in the temporal pole. Posterior to the splenium this band of cortex was notched by a fissure (*? c.f.*) which, from the arrangement of the cortical arteries, seemed to represent the anterior limb of the calcarine fissure.

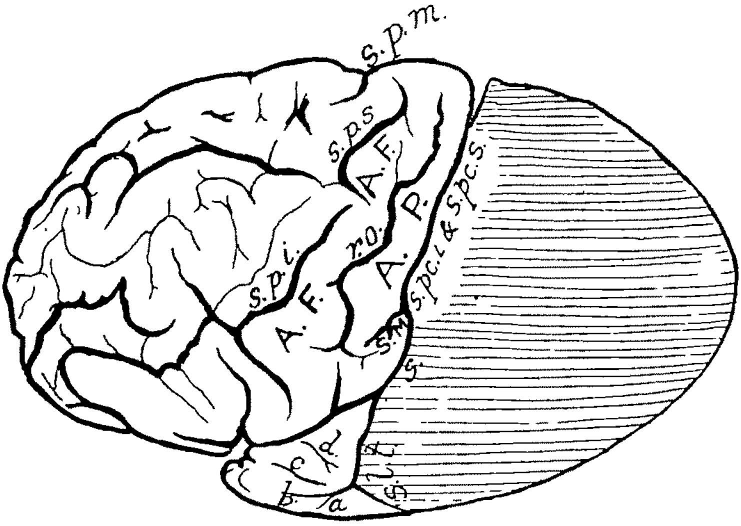


FIG. 3.

External aspect of Left Hemisphere (reduced from a drawing. Cyst wall shaded).

A.F., ascending frontal convolution. *A.P.*, ascending parietal convolution. *S.M.*, anterior limb of the supramarginal convolution.

s.p.m., sulcus præcentralis medialis. *s.p.s.*, superior præcentral fissure. *s.p.i.*, inferior præcentral fissure. *r.o.*, fissure of Rolando. *s.*, upturned end of fissure of Sylvius joining *s.p.c.i. and s.p.c.s.*, the inferior and superior postcentral fissures. *s.l.t.*, fissure separating remnant of temporal lobe from cyst.

a b c d mark the four shallow fissures on the remnant of the temporal lobe.

Above the level of this notch the cyst was separated from the cortex by a comparatively deep fissure—the continuation of the subparietal—below, however, the cortex gradually shelved into cyst wall, but the limits of cyst and cortex were approximately defined by a very shallow fissure (*s.s.d.c.*)

Behind this shallow fissure the cyst wall was indeed, for three-quarters of an inch, slightly thickened and its outer surface was granular owing to a thin granular layer of cortex, but the thickness was such that the wall was still quite translucent, and the shadow of a finger could be seen through it.

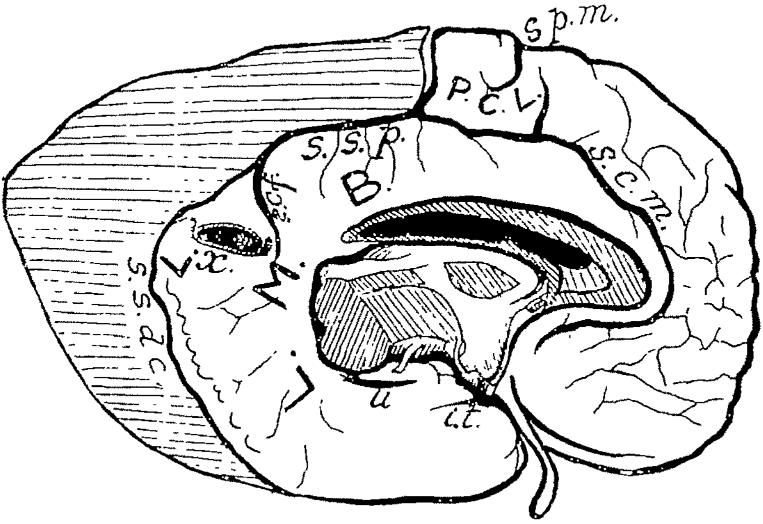


FIG. 4.

Inner aspect of Left Hemisphere after removal of hind brain (reduced from a drawing. Cyst wall shaded).

P.C.L., paracentral lobule. *L ?* portion of lingual lobule. *L.M.B.*, limbic lobe.

s.p.m., sulcus præcentralis medialis. *s.c.m.*, callosomarginal fissure, the upturned end of which separates *P.C.L.* from the cyst. *s.s.p.*, subparietal fissure. *? c.f.*, probably the anterior limb of the calcarine fissure. *u.*, the uncus. *i.t.*, incisura temporalis. *s.s.d.c.*, shallow fissure defining cyst from thinned-out cortex anterior to it.

.c., stalk of cystic diverticulum, which lay over cerebellum.

On the Left Side.

On the left side the similarity between the cyst and that on the right in its limits, and more especially its mode of origin, was most remarkable. The only differences were that, on the inner aspect of the left hemisphere (fig. 4), the upward turn of the calloso-marginal fissure (*s.c.m.*) formed the posterior limit of the thick cortex above the limbic lobe, so that there was on the left side no precuneal lobule anterior to the cyst, and, further, there was present on the left side *a cyst springing as an offshoot from the main cyst and lying over the cerebellum.*

This cyst was an evolution from the thinned cortex of the limbic lobe immediately below the notch representing the anterior limb of the calcarine fissure (*? c.f.*)

At its origin it communicated with the left ventricle by an opening of oval shape 1½ cm. in longest diameter, and from this narrow stalk (figs. 4 and 5, *x*), widened out into a roughly triangular body which had insinuated itself under the tentorium, the arachnoid with which it was covered being firmly adherent to the membranes in its neighbourhood. The anterior and outer half of its upper surface was of a yellow-white colour, due to a very thin layer of cortex, but the rest of the cyst was of a bluish colour and extreme tenuity.

With these exceptions the constitution of the main cyst wall on the left side and its mode of origin from the cortex were exactly similar to those found on the right.

(2) *Macroscopic Appearance of the Cortical Remnant.*

Having thus defined the limits of the cyst it were best to describe the appearance of the thicker cortex and to enumerate the convolutions thereon before defining the actual portions of the cortex destroyed.

On the Outer Surface of the Right Hemisphere (fig. 1).

Of the parts above the Sylvian fissure, the whole of the *frontal lobe* was present and all the ordinarily named fissures and convolutions could be traced. Although the complexity of the convolutions was normal or even greater than usual, yet the convolutions were of small size; thus the ascending frontal (*A.F.*) averaged 11 mm., its greatest breadth being 16 mm., the superior frontal averaged 23 mm., the middle 30 mm., and the inferior, or Broca's, 32 mm. Besides this microgyria there was considerable flattening; thus the Rolandic fissure by the genu was only 15 mm. in depth. The fissure of Rolando ran into the superior postcentral sulcus 18 mm. from the supero-mesial border

The *parietal lobe* was represented by the ascending parietal convolution (*A.P.*), which was extremely small, its broadest part, opposite the second genu of Rolando, being 10 mm. broad, and its lower part being a mere ribbon averaging 5 mm. There was also a small piece of supramarginal cortex, 23 mm. in extent and 8 broad, corresponding (fig. 1, *S.M.*) to the anterior limb of the supramarginal convolution.

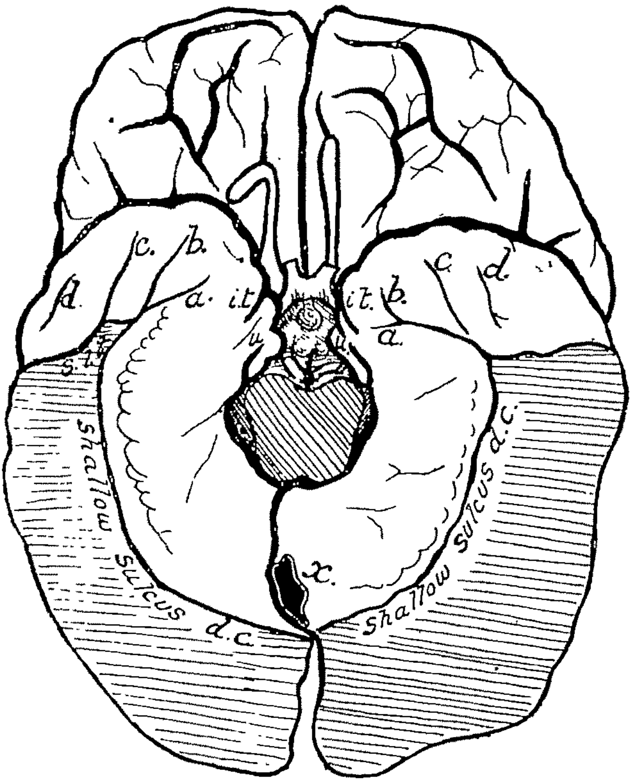


FIG. 5.

Inferior aspect of the cerebrum, after removal of hind brain (reduced from a drawing. Cyst wall shaded).

a b c d, the four shallow fissures on the remnant of the temporal lobe.

s.s.d.c., shallow fissure defining cyst from thinned-out cortex internal to it. *s.l.t.*, fissure defining remnant of temporal lobe. *i.t.*, incisura temporalis. *u.*, uncus

x., the stalk of the cystic diverticulum from the left main cyst, which lay upon the cerebellum.

The island of Reil was fully developed in proportion to the frontal lobe, and its surface showed all the usually described convolutions and fissures.

The remnant of the temporal lobe merits, in view of the localisation of hearing, a detailed description. As may be seen in figs. 1 and 5, it was roughly of U or V shape, the point of the V corresponding to the temporal pole. The two limbs of the V were separated by a well-marked fissure (*b*). Of these limbs the inner formed the inferior extremity of the strip of cortex representing the limbic lobe, and was in breadth by the uncus 4½ cm., and immediately behind the uncus 3 cm.; the outer limb represented the remnant of such parts of the temporal lobe as are found on the external aspect of the hemisphere, and had two surfaces—an upper or opercular (not exposed in the figures) and an outer or exposed. The opercular surface was divided into convolutions by a fissure running parallel to the limiting sulcus of the isle of Reil; and the convolution adjacent to the isle was greatly thinned. The outer surface, triangular in shape (*c* and *d* in figures lie on this outer surface), was 5 cm. long from apex to base (the fissure *b*), and 2½ cm. broad at this base.

The remnant of the temporal lobe on the left side (figs. 3 and 5) may be conveniently described here. It only differed from the right in that on its opercular surface (*vide* plate IV.) the convolution adjacent to the island of Reil was not so much thinned, and the outer surface measured 6½ cm. by 3½, as compared with 5 cm. by 2½ on the right.

It was impossible to determine the exact significance of the fissures (*a*, *b*, *c*, *d*); *d* was hardly visible and obviously represented no named fissure, *c* and *b* corresponded in position with the anterior extremity of the parallel or first temporal fissure in normal brains, and *a* pos[illegible] represented the anterior end of the collateral fissure.

The cortex forming the convex borde[illegible]mnant of lobe was thick, but rapidly thinne[illegible]iorly towards the cyst, and also, as we have se[illegible]wards the insula; under these thinned walls t[illegible]ity of the lateral ventricle extended towards the [illegible]le.

This cortical remnant represents, then, at most the temporal pole with the uncus, the anterior or lower two-thirds of the superior temporal convolution with the temporal operculum, perhaps the tip of the middle and inferior, and possibly a strip of the occipito-temporal convolutions.

On the Inner Aspect of the Right Hemisphere (fig. 2).

The frontal lobe save for microgyria and flattening was in every way normal, all the known fissures and convolutions being easily made out. Behind the paracentral lobule on this side of the brain lay a portion of the *precuneus*, probably the anterior third, if the parietal and occipital fissure may be located by the small parieto-occipital artery seen running over the cyst wall in this region.

As far as the start of the anterior limb of the calcarine fissure (? *c.f.*) the limbic lobe was also relatively normal; but the band of cortex which curved round from this point to the temporal pole was thick at its anterior concave border and grooved by the dentate fissure, but rapidly thinned out posteriorly towards the cyst. There was no sign of the collateral fissure unless it was represented by the shallow fissure (*s.s.d.c.*) which defined the cyst from cortex.

This band of cortex represents, then, the isthmus, a very small piece of thinned-out lobulus lingualis, the hippocampal convolution also greatly thinned and possibly a narrow strip of occipito-temporal convolution. The hippocampus and fascia dentata are well formed.

On the Inferior Aspect of the Right Hemisphere (fig. 5).

The orbicular surface of the frontal lobe showed all the named fissures and convolutions; the temporal lobe seen in this view has already been described.

[illegible]*ft Hemisphere* (figs. 3, 4 and 5).

T[illegible]phere was almost identical with the right. The c[illegible] difference lay in the entire absence of the precu[illegible]e protrusion of the thinned-out lingual the thinned-out portion of the limbic lobe; portions were also taken from the thin and thick parts of the wall of the cyst which lay over the cerebellum.

These were fixed in 5 per cent. formalin, in Müller following 5 per cent. formalin, and according to Weigert's chromic acid method for glia staining.

Typical portions of the cyst wall were found to be lined by ependyma and covered by pia-arachnoid and to be composed chiefly of glia fibres running for the most part parallel to the surfaces, among which were cells, numerous corpora amylacea, occasional medullated fibres, and small vessels. A detailed examination was made of each of these constituents.

The pia-arachnoid was found upon the cyst wall, as upon the cortex, to be of healthy appearance, the vessels being normal and showing an unchanged elastic lamina after Weigert's elastic stain.

The ependyma consisted everywhere of very much flattened oblong cells. In preparations stained by Weigert's glia method, neutral red or toluidin blue, the ground substance stained opaquely, and was obviously degenerated; whilst in most cells large oval nuclei could be made out, in others they were indistinguishable. The nuclei for the most part stained faintly, some stained as a homogeneous dark mass. No cilia could be made out, and no ciliary basal bodies.

The degenerated cells did not give any amyloid reaction.

The glia.—The cyst wall was composed mainly of glia fibres; these ran for the most part parallel to the cyst surface and stained well by Weigert's glia method. Besides glia cells, which stained normally, others showed signs of degeneration, some being swollen and having the chromatin collected into a mass, others staining uniformly dark and opaque.

Medullated fibres.—Sections cut 30 μ thick and stained by the Kultchizky-Weigert-Pal method showed the presence of medullated fibres which varied in number in the different parts of the cyst wall.

In the thin portion of the cyst, which lay over the cerebellum, only a very occasional single fine fibre could be found; in the thicker portion more numerous fibres—up to fifteen running parallel—could be found in one section.

In the wall of the main cyst, where not specially thickened, here and there portions of fibres could be seen running longitudinally, but the largest number of parallel fibres seen at one place was six.

In the thickening seen in the wall, as it sprang from the bottom of the deep fissure behind the ascending parietal convolution, the fibres were more numerous, eight to twelve parallel fibres running throughout.

Where the posterior boundary of the limbic lobe was only approximately defined from the cyst by a shallow limiting fissure (figs. 2 and 4, *s.s.d.c.*), large numbers of fibres could be seen running longitudinally beneath the thin cortex anterior to the fissure and radiating into the cortex; posterior to the fissure these gradually became less in number until, at the distance of about one centimetre, only some eighteen fibres could be focussed, all now running longitudinally.

The corpora amylacea.—In all portions of the cyst wall examined, round bodies were seen which stained a mahogany brown with Lugol's iodine, changed to dirty slate blue by dilute sulphuric acid; treated with methyl gentian violet, hydrochloric acid, and potassium acetate, they stained a beautiful pink—a colour which they also assumed after staining in Unna's polychrome methylene blue and differentiating with Unna's mixture.

These bodies were seen chiefly at the surfaces next the ependyma and membranes, but were by far the most numerous under the ependyma. They could also be seen in places surrounding the small vessels in the cyst. Perfectly round in shape, without any signs of laminæ, they were for the most part about the size of an average pyramidal cell, though many smaller ones of the size of the swollen ependyma cells could be seen. The most careful search, however, failed to show any proof of transition from cells into such bodies. It may here be stated that no such bodies were found in the cortex proper, save in the ascending parietal convolution at its base opposite the origin of the cyst, and one or two close to its outer surface.

thickenings behind the fissures limiting the temporal and lower part of the limbic lobes, was devoid of cells capable of exercising the functions of cortical tissue.

(2) *Microscopical Examination of the Cortex.*

Portions were taken from the ascending frontal and parietal convolutions, on the left side opposite the mid-frontal convolution, and on the right side oposite the inferior frontal convolution (*i.e.*, anterior to letter *M* in fig. 1). Pieces of cortex were also taken from the thinned portion of the limbic lobe on the right and from the remnant of the temporal lobe on the left side.

These were fixed in 5 per cent. formalin, Muller's fluid after formalin, and according to Weigert's glia method.

The glia was normal in amount and structure.

Nerve cells.—The relative number and histological characteristics of the nerve cells in the ascending frontal and parietal convolutions were normal, except in the portion of ascending parietal taken from the left side. From the base of this portion the cyst had origin (*i.e.*, between letters *A* and *P* in fig. 3), and of its cross-section, the posterior and lower third opposite the cyst origin was devoid of nerve cells, being in structure similar to the cyst wall itself.

The proportion of large to small pyramidal cells was similar to that in control preparations.

The cortex from the limbic and temporal lobes, although greatly thinned, contained large numbers of healthy pyramidal cells.

The medullated fibres.—Sections of the ascending frontal and parietal convolutions stained by the Kultschizky-Weigert-Pal method were found to differ from similar sections of healthy cortex of equal thickness.

In Edinger's "Intra-radial felt-work" below Baillarger's band there was a very marked diminution in the number of tangentially-directed fibres. The number of radiating columns was similar to that in the healthy brain, but there was a great diminution in the number of individual fibres in each column, and this diminution was due to a loss of fine fibres. Thus, in sections cut 20 μ thick, the columns in the healthy brain contained, on an average, two to three coarse and eight or more fine, whereas those of the patient's brain two to three coarse and, at most, two fine.

In the "supra-radial felt-work" there were hardly any tangential fibres seen, and there was a great reduction in the number of the "true tangential fibres" of Exner's layer.

V. — MACROSCOPIC AND MICROSCOPIC EXAMINATION OF PARTS OF THE CENTRAL NERVOUS SYSTEM OTHER THAN THE CYST AND CORTEX.

In the following description only relative abnormalities in size will be mentioned. The basal ganglia were thought in the autopsy to be absolutely smaller than in healthy adult brains; when the following examination was made the brain had been so long exposed to strong formalin that measurements of absolute size were valueless.

The Veins.

The veins of Galen and their tributaries were normal; the veins over the cyst walls were much smaller than over the rest of the cortex.

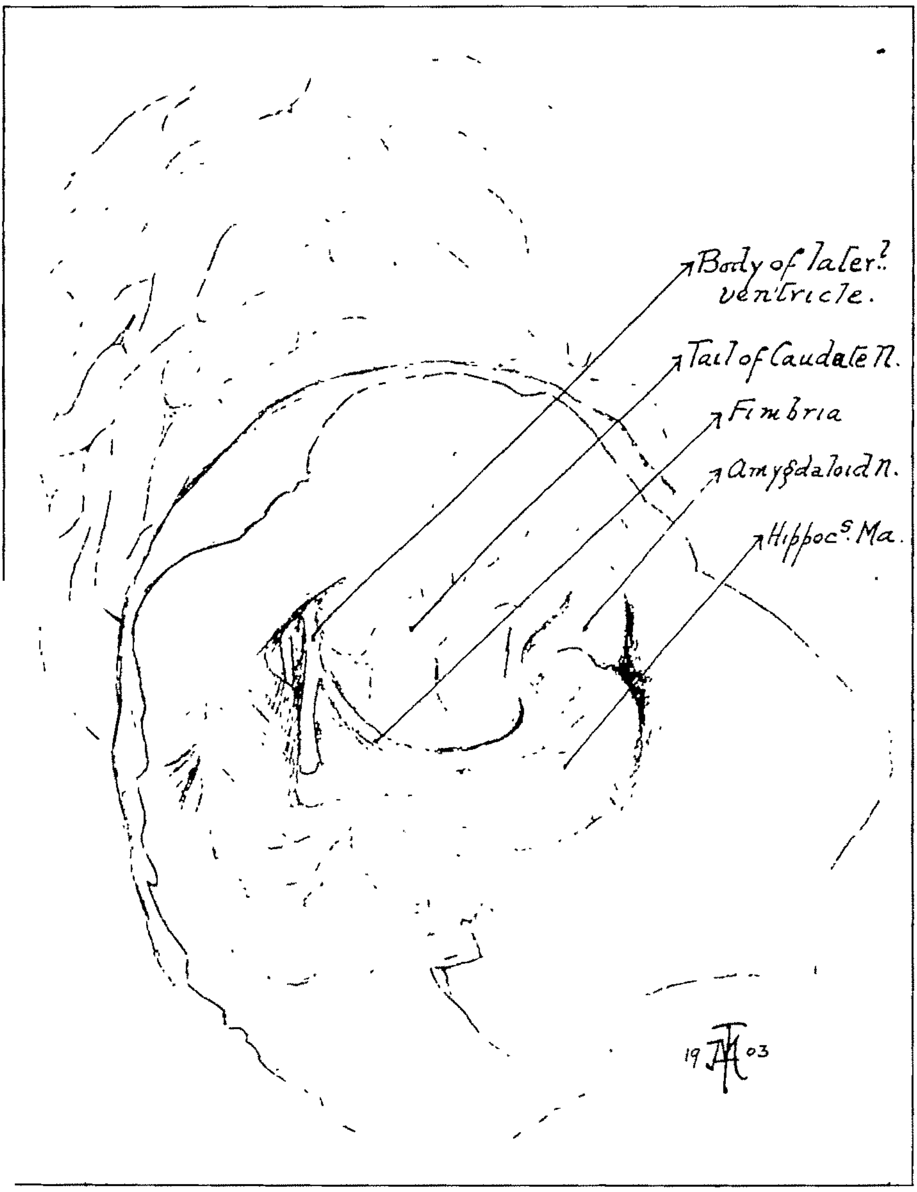
Cerebral Arteries.

The posterior cerebral arteries were from two-thirds to one-third the calibre of the anterior and middle, and their terminal calcarine and parieto-occipital branches could be seen as very slender arteries running on the cyst wall. The named branches of the anterior and middle cerebrals could all be traced, but such as reached the cyst area immediately became of minute calibre.

The Ventricles.

Both the *lateral ventricles* were greatly dilated, the anterior horns being considerably widened and having a depth of 3½ cm., whilst the posterior and descending horns (plate III.) had been expanded into one cavity—the cyst

PLATE III.



View of interior of right cyst, seen on looking into the dilated posterior horn and descending horn of the right ventricle, through a window formed by the removal of the posterior or occipital portion of the cyst wall.

(*From a drawing--natural size.*)

proper—to which the hind part of the corpus striatum formed an almost perpendicular anterior wall.

The left ventricle had been subjected to rather more pressure than the right, as was shown not only by the extension of the supracerebellar cyst, but by the bulging across the middle line of the cortex adjacent to the origin of this cyst.

On the left side the retiring angle between the head of the caudate nucleus and the anterior extremity of the optic thalamus was roofed in by a membrane passing to the fornix, whilst the adjacent part of the caudate and optic thalamus showed a slightly raised white patch of considerable extent, due probably to old inflammatory thickening. This membrane was continuous with the upper layer of the choroid, and had an oval aperture which led into a closed cavity, the floor of which was formed by the adhesion of the deep layer of the choroid to the fore part of the optic thalamus and fornix. Beneath this in the fore part of the optic thalamus was a small cyst containing fluid; the upper wall of this cyst was found on microscopical examination to consist of degenerated badly-staining substance, in which an occasional medullated fibre could be seen; its smooth internal surface bore no trace of ependyma.

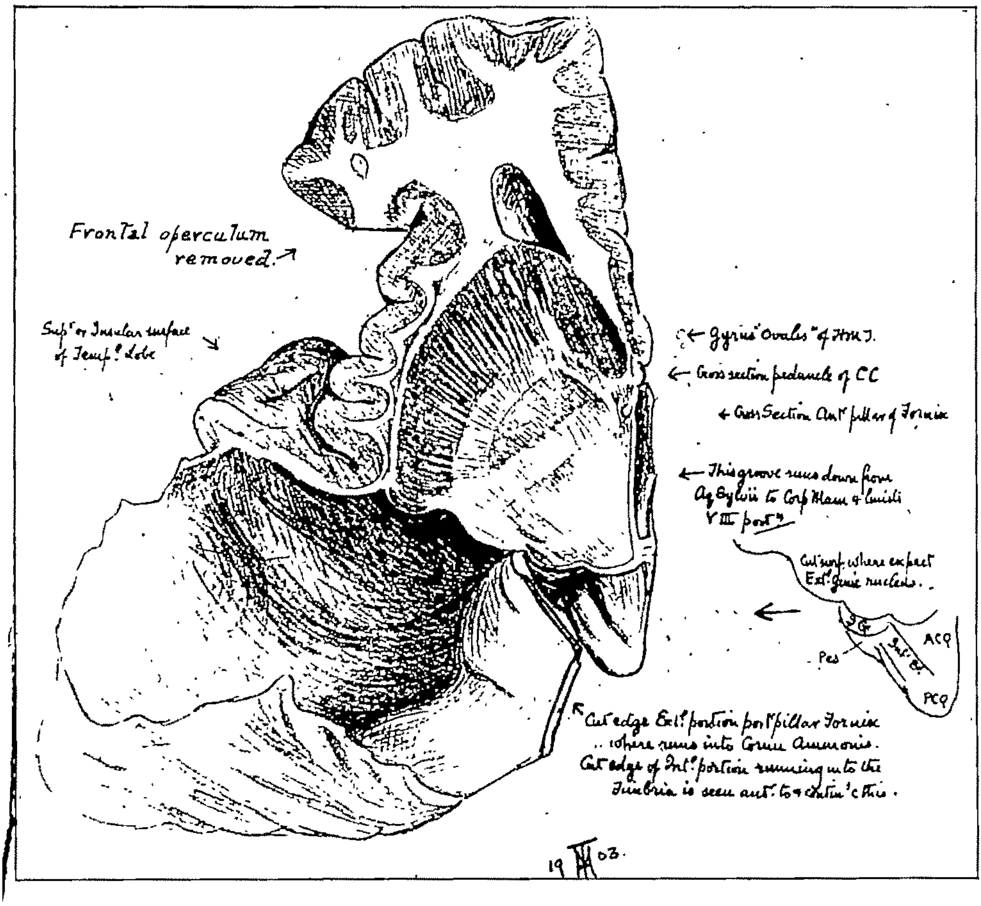
There was thus no connection between the third ventricle and the left lateral ventricle, a probe passed through the left foramen Monro merely impinging on the deep layer of choroidal membrane. The left wall of the *fifth ventricle* was dragged over by these adhesions.

It is to be regretted that in examining the veins of Galen the velum interpositum was torn from the neighbourhood of the right foramen Monro. There was no cyst in the head of the right optic thalamus and the only abnormality seen was a thickening in the angle between the head of the caudate nucleus and anterior extremity of the optic thalamus similar to that on the left side.

The lateral part of the body of the *fornix*, the posterior pillars and fimbriæ, had all been flattened out by the intraventricular pressure.

The cavity of the *third ventricle* was distended and the

PLATE IV.


Frontal operculum removed.
Supr or Insular surface of Templ Lobe
Gyrus "Ovalis" of HMJ.
Cross section peduncle of CC
Cross Section Antr pillar of Fornix
This groove runs down from Aq Sylvii to Corp Mam & limits V III postr
Cut surf. where expect Extl Genic nucleus.
IG
Pes
Inf. Br
ACQ
PCQ
Cut edge Extl portion postr pillar Fornix .. where runs into Cornu Ammonis. Cut edge of Intl portion running into the Fimbria is seen ant. to & contin' c this.
19 III 03.

Horizontal section, left hemisphere, showing condition of retrolenticular part of internal capsule.

(From a drawing—slightly reduced.)

others stained by the Weigert-Pal method, showed no signs of degeneration.

The constitution of the retrolenticular and sublenticular portions of the internal capsule was examined in Weigert-Pal preparations of serial vertical sections extending through the entire brain on the right side as far forward as the temporal pole.

From the stratum zonale, and to a less extent from the outer surface of the posterior part of the optic thalamus, fibres passed downwards and slightly forwards to reach a position above and to the inner side of the bundle of fibres representing the transversely cut lower end of the tænia semicircularis; here they were joined by a small "bundle of Türck," and running forwards beneath the lenticular nucleus apparently ended in the cortex of the temporal pole. These fibres, which correspond to the temporo-thalamic fibres of Arnold,[1] formed with the small bundle of Türck the sublenticular portion of the internal capsule, which was smaller than normal.

In the *retro-lenticular* portion of the internal capsule a few fibres passed from the outer surface of the optic thalamus outwards and upwards towards the postero-superior angle of the insula. Fibres from the cerebral peduncle also passed in the upper part of this portion of the capsule to reach the cortex above and behind the insula.

As will be found below in the description of the central connections of the optic nerve, no fibres passed from the external geniculate ganglion into the retro-lenticular portion of the capsule. There was indeed in this portion of the capsule entire absence of fibres passing from the pulvinar thalamus, external geniculate and anterior corpus quadrigeminum, in the direction of the occipital, lingual, fusiform and posterior part of the parietal lobes, as was to be expected considering the cystic destruction of these lobes, and the absence of any traces of "optic radiations" (Gratiolet), or indeed any "stratum sagittale externum" (Sachs), in the cystic outer wall of the posterior and inferior horns of the lateral ventricle.

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[1] Dejerine, Anatomie des Centres Nerveux, Tome II., 1901; pp. 41-47.

"*Türck's*" *bundle* (the "temporal cerebro-cortico-pontal path") could be easily traced, as the fibres were more easily decolourised than the other peduncular fibres. In the peduncle these fibres formed the most lateral bundle, occupying at most only one-ninth of the entire cross section. At a level anterior to the termination of the atrophied external geniculate, and posterior to the posterior extremity of the lenticular nucleus, Türck's bundle left the other peduncular fibres to join the thalamo-temporal fibres and form the sublenticular portion of the internal capsule.

It may here be stated that in the anterior sections of this series of vertical sections the *inferior peduncle of the optic thalamus* was seen to be normally developed.

The Mesencephalon and Central Connections of the Optic Nerve.

(*a*) *Macroscopically.*

The optic nerves were seen on the base of the brain to be relatively small, their diameter on cross section being only slightly larger than that of the third cranial nerve.

The nerve tracts were also small, and a lateral root could only be detected with difficulty.

The external geniculate bodies could barely be recognised as small and but slightly projecting oval bodies having a surface area less than two-thirds of that of the well-marked and projecting internal geniculate.

The superior and inferior brachia were both well marked and in due proportion to the rest of the brain.

The anterior and posterior corpora quadrigemina had been flattened out by the pressure of the overlying cysts, but seemed to be of relatively normal proportions.

In sections through the mesencephalon the relation in size of the crura to the tegmentum seemed abnormal, the crus not extending laterally so far as in normal controls.

(*b*) *Microscopical.*

Microscopical preparations were made of the intracranial portion of the right optic nerve in transverse section, the right half of the chiasma with the end of right nerve and start of the right tract in horizontal section, and the left half in sagittal transverse section, and the right optic tract before reaching the peduncle in vertical transverse section. Control sections were cut in an exactly similar manner from a healthy brain.

The posterior part of the optic thalamus, the internal and external geniculates and the anterior corpora quadrigemina were cut in serial sections roughly parallel to the roots of the optic tracts.

These sections were stained by the Kultschizky-Weigert-Pal method, neutral red being used as a counterstain for the examination of the cells.

The intracranial portion of the right optic nerve was seen in the transverse section to be of minute size and flattened from above down.

Between the sheath of membranes and the central nerve bundles was a continuous sheath consisting of a network of glia fibres divided into imperfect loculi by trabeculæ reaching from the pia mater. This sheath resembled in structure, therefore, the subpial glia sheath described by Fuchs (1881)[1] and seen in the normal nerve, but it was absolutely very much larger, and relatively it occupied about one-sixth of the total diameter of the portion of nerve within the membranous sheath. It was apparently of larger size on the dorsal and inner aspects of the cut surface, and in the portion adjacent to the central nerve bundles could be seen scattered fibres stained by the Weigert-Pal method.

The central portion within this glial sheath consisted of nerve bundles. These bundles were very small and each contained far fewer fibres than in the normal nerve, the individual fibres appearing more isolated from each other. The nerve fibres were of different calibre, but were almost all very small, the largest being smaller than the numerous large fibres in the control.

There was no sclerotic thickening in the nerve.

The horizontal section of the chiasma was very much smaller than the control. The pressure within the third ventricle had hollowed the chiasma so as to form a thin lamina bulging ventrally, and it was therefore impossible in any one section to cut the entire chiasma horizontally. The sections showed that the fibres from the nerve in part passed direct into the corresponding tract and in part crossed in the chiasma; it was not possible from these sections to determine whether there was any tract of Gudden, and it may be here stated that in the control sections no definite strand of fibres could be seen passing from one tract to the other in the posterior concavity of the chiasma.

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[1] "Encyclopédie française d'ophthalmologie," 1903, p. 688.

Sagittal transverse sections through the chiasma.—In these sections the bundles constituting *Meynert's commissure* were seen, and, being of size and appearance similar to those in the controls, they rendered the atrophy of the chiasma very conspicuous.

The chiasma was flattened and in area was reduced to at least one-tenth of the control. The fibres were for the most part small, the largest (smaller than the large fibres in the control) being found chiefly at the posterior end.

Transverse sections through the right tract.—The tract was not more than one-tenth of the control, the larger fibres being found chiefly in a small group at the posterior end and extending thence through the centre and coming to the exposed or ventral surface towards the anterior end. In the normal tract it was not possible to determine any preponderance of large fibres at any one spot.

Meynert's commissure was normal.

On comparing the section of the optic nerve with that of the tract no accession of fibres to the tract could be recognised, such as would be expected if a true commissure of Gudden existed in man.

In these sections of the chiasma and tract, as in the optic nerve, no sclerotic thickening could be detected.

The Central Nuclei.

The external geniculate was of exceedingly small size and showed no traces of its usual lamellar arrangement. The only cellular nucleus was a mass which, at its largest, was

2½ mm. by 1½ mm., most of the cells being collected into a small kidney-shaped group filling the inferior third of this area.

This cellular nucleus was only seen anterior to the beginning of the internal geniculate, and it extended for only a very short distance.

The cells were of pear shape and averaged 16 by 9 μ in size.

The internal geniculate was of relatively normal size, being in greatest area 8 mm. by 4 mm., and contained very numerous cells, averaging 25 by 14 μ.

The pulvinar examined microscopically showed no signs of atrophy and contained numerous cells, the largest of which were about 30 by 16 μ.

The stratum zonale was well marked.

The anterior corpus quadrigeminum did not show any recognisable atrophy and, in cross section, was in normal proportion to the posterior corpus quadrigeminum. The cellular content did not appear to differ from the normal with which it was compared. In the deepest layer a few giant cells, measuring 60 by 34 μ, were seen, but most of the larger cells averaged 26 by 14 μ.

The arrangement of cells and fibres was similar to the normal, and from the sections it was not possible to state that the fibres in any of the three fibre zones were reduced in number.

Central Distribution of the Optic Tract.

In serial sections through the optic thalamus and the anterior corpus quadrigeminum two main sets of fibres were met with, *i.e.*, the fibres of Meynert's commissure and the fibres of the true optic tract.

The fibres of Meynert's commissure could be readily separated from the other fibres of the tract. Closely applied to the surface of the peduncle, lying between the peduncle and the optic fibres, and curving round the outer angle of the pes, they swept round and through Türck's bundle in the direction of the Corps de Luys, many of the fibres being found in sections anterior to the level at which the true optic fibres were first met. The fibres of Meynert's commissure were calculated to represent about one-third of the total number of fibres passing round the peduncles, and from this an idea of the atrophy of the optic tract fibres may be gained.

The foremost *fibres of the true optic tract* were found posterior to the uppermost fibres of Meynert's commissure. Most anteriorly they were represented by a few fibres separated widely from each other and running towards the external geniculate; apparently, however, they did not end in this ganglion, but seemed to be part of the fibres next met with. These were several small bundles of fibres which ran through the external geniculate and upwards along the external surface of the pulvinar. At a slightly lower level the sections showed the kidney-shaped cellular nucleus of the external geniculate, and the outer surface of this small nucleus was covered by a well-marked fibrous lamina formed by fibres, now in considerable numbers, passing from the tract along the outer surface of the nucleus to run vertically upwards to the outer surface of the pulvinar.

These vertical fibres were joined by fibres which started in, or ended in, the outer and upper part of the nucleus. No fibres could be found passing from the tract or the external geniculate in an outward direction corresponding to that of the optic radiations in control brains.

The course of the remaining fibres was more difficult to follow. Some passed anterior to the internal geniculate to reach the anterior corpus quadrigeminum, the remainder passed beneath and posterior to the internal geniculate to reach the outer aspect of the posterior brachium. The sections did not show any fibres definitely ending in the external geniculate. Although the fibres which reached the outer aspect of the posterior brachium appeared to pass on to join the anterior brachium, yet it was impossible to be certain that they did not join the upper and outer part of the posterior brachium.

From comparison with sections of normal tissue it was impossible to state definitely that there was any atrophy in the *anterior brachium.*

The *posterior brachium* was at any rate mainly formed by large bundles of fibres passing from the internal geniculate, and was undoubtedly normal in size.

To Summarise, therefore, the Changes met with in the Optic Nerve and its Connections.

There was a very great diminution in numbers of fibres in the nerve, this diminution was unaccompanied by sclerosis, and the excess of subpial glia sheath suggested a non-development rather than an atrophy of fibres. The fibres that remained were of small calibre.

The chiasma and tracts showed a corresponding loss of fibres.

The external geniculate was extremely atrophied and no fibres could be traced into it from the nerve.

The pulvinar and anterior corpus quadrigeminum received all the fibres of the tract save perhaps some which passed to the posterior corpus quadrigeminum in the posterior brachium. The anterior corpus quadrigeminum was apparently not atrophied, and the pulvinar, though it appeared macroscopically to be deficient posteriorly, on microscopic examination showed no undoubted signs of degeneration.

The intracerebral path of the optic nerve (optic radiations) was entirely wanting.

Meynert's superior commissure was unaltered.

Hind Brain.

No abnormalities were seen in the pons and fourth ventricle.

The superior vermis of the cerebellum, instead of being convex, was depressed so as to form the central portion of an oval depression in which lay the supracerebellar cystic offshoot from the main cyst of the left hemisphere.

All the lobes and lobules of the cerebellum were present, and no abnormality could be detected on section.

No adhesion could be found in the roof of the fourth ventricle.

The Medulla and Cord.

The medulla was normal.

The cord measured 14⅜ inches from the end of the conus medullaris to the start of the filum terminale.

The cord was placed immediately after removal into Müller's fluid and sections were taken from the upper and lower cervical, mid-dorsal, lumbar and sacral regions and stained by Weigert-Pal's method. These sections were of good shape and of larger size than control specimens from a girl of 24, and no abnormalities could be detected.

The cells in the cervical and lumbar enlargements, stained with toluidin blue, were of good shape with healthy nucleus and contained Nissl's granules.

(*C*) REMARKS.

The abnormalities found in the above examination of the central nervous system consist in brief of a distension of the ventricles and destruction of a large area of the cortex, accompanied by certain changes in the remaining cortex, in the corpus callosum, in the retro- and sub- lenticular portions of the capsule, and in the optic nerve and its central connections.

The facts settled beyond dispute in the study of the interdependence of parts of the central nervous system justify the statement that the changes in the corpus callosum, the capsule and the optic nerve connections were undoubtedly secondary effects of the cortical destruction, and suggest a similar explanation of the abnormalities in the thick cortex.

It will be most convenient to consider each secondary change in turn before discussing the pathology of the primary cortical destruction.

VI.—Changes Secondary to the Primary Cortical Lesion.

In the portions of thick cortex which were examined microscopically, whilst the number of coarse fibres in the radiating columns was normal, a deficiency was found in the fine radiating fibres and also in the tangential fibres. Now, although there is no direct evidence in the literature that such a change is secondary to cortical destruction, yet it is not unreasonable to suppose that commissural fibres are of finer calibre than the projection fibres of the pyramidal tract, and that this deficiency was caused by a loss of the commissural fibres which would have united this thick cortex with the extensive cortical area destroyed.

The corpus callosum.—Accompanying the destruction of the whole of the occipital lobe except a minute fragment of lingual lobule, of all the angular convolution and of all save the most anterior strip of the parietal cortex, the posterior part of the body of the corpus callosum was found to be much reduced and the splenium was markedly atrophied.

In the study of degenerations following cortical destruction it has been shown that the parietal lobes are connected through the hinder part of the corpus callosum, whilst in the splenium Dejerine has shown that fibres pass from the cuneus, occipital pole and lingual and fusiform convolutions,[1] and Ferrier and Turner[2] have found in the monkey fibres also from the external occipital convolutions and from the angular gyrus.

The atrophy therefore of the hinder part of the corpus callosum, found in the present case, agrees with observations already made. The fact, however, that the atrophy was not complete furnishes further information concerning the construction of this portion of the corpus callosum.

Other fibres than those from the occipital, angular and parietal cortex passed in the splenium and hinder part of the body of the corpus callosum and these could only be fibres passing to or from the limbic lobes, as was indeed proved by dissection in the case of the splenium.

The sublenticular portion of the internal capsule and Turck's bundle.—The sublenticular portion of the internal capsule in the normal brain consists of two bundles passing to the temporal lobe, the bundle of Türck and the temporo-thalamic bundle of Arnold.[1]

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[1] "Contribution à l'étude de la dégénérescence des fibres du corps calleux." M. et Mme. J. Dejerine. *Société de Biologie* (Compt. rend.), 1892, p. 579.

[2] Ferrier and Turner. *Philosophical Transactions*, 1898, B. p. 9; *vide* also Vialet on Corpus Callosum, "Les Centres Cerebraux de la Vision." Thèse de Paris, 1893.

In the present case there was some atrophy in the sub-lenticular portion of the internal capsule, and this was due in part at any rate to a diminution in the size of Türck's bundle. This diminution in Türck's bundle explains also the reduction noted in the size of the cross section of the crura of the mesencephalon.

Türck's bundle was not, however, completely atrophied, but was represented by fibres filling about one-ninth of the cross section of the cerebral peduncle, and these fibres were traced to the temporal lobe. This lobe, as we have seen above, was represented by at most the temporal pole with the uncus, the anterior two-thirds of the superior temporal convolution with the temporal operculum, perhaps the tip of the middle and inferior, and possibly a strip of the occipito-temporal convolutions.

Türck's bundle is known to consist of fibres which connect the cerebral peduncle with the temporal lobe and degenerate in a descending direction, but there has been considerable controversy as to its exact origin. According to Dejerine[2] it has origin from "the middle portion of the temporal lobe, and in particular the cortex of the second and third temporal convolutions," whilst Flechsig believes that it has origin from the auditory sense area in the gyrus temporalis superior and the gyri temporales transversi. Mills and Spiller report a case which strongly opposes the view that the origin is from the superior temporal convolution, though Ferrier and Turner found secondary degeneration in the lateral bundle of the pes after experimental extirpation of this convolution in the monkey.[3]

Unfortunately the loss of fibres in the present case can be explained as due to the destruction either of the upper one-third of the superior temporal convolution favouring Flechsig's view, or of the middle and inferior convolutions favouring Dejerine, whilst, although it seems probable that the remaining fibres had origin in the remnant of the superior convolution and operculum, yet there was no evidence that the origin was not in the cortex forming the temporal pole.

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[1] Dejerine. *Anatomie des Centres Nerveux*, Tome II., 1901, p. 41.

[2] Dejerine. *Loc. cit.*, Tome II., p. 79, and *infra*.

[3] Barker. "The Nervous System," 1900, p. 1044, where this question is discussed.

The retro-lenticular portion of the internal capsule was extremely atrophied. The few fibres found in it could be traced from the outer surface of the optic thalamus to the temporal operculum and the posterior angle of the insula.

Where the retrolenticular portion of the capsule was continuous with the posterior limb there were found also some of the most posterior of the peduncular fibres passing to the cortex above and behind the insula.

In the normal brain it has been shown that the retrolenticular portion of the internal capsule is formed by a mass of fibres of different anatomical and physiological significance, but all included in the term "the optic radiations of Gratiolet." The middle band seen in the external sagittal layer of the wall of the lateral ventricle and known as the "optic radiations," is constituted in its posterior part by fibres springing exclusively from the occipital cortex and the lingual and fusiform lobes, but as it approaches the ganglia of the base it is constantly increased by fibres from the parietal and temporal lobes. Thus formed it passes into the retrolenticular portion of the capsule, and then breaking into numerous bundles it ends in the optic thalamus, the external geniculate, the anterior corpora quadrigemina and the internal geniculate.

Amongst these fibres are the true visual fibres from the cortical visual area, which from the now large mass of clinical and experimental evidence undoubtedly lies within the limits of the cortex of the cuneus, lingual and fusiform lobes, and possibly the outer surface of the occipital lobes and the angular gyri. The exact localisation of the centre of vision within these limits is of no importance in the consideration of the present case.

The true visual fibres are connected with the pulvinar, the external geniculate and the anterior corpora quadrigemina. von Monakow has shown experimentally that the internal geniculate is connected with the temporal lobe and is part of the mechanism of hearing and not vision.[1]

The lesion found in the present case is in strict accordance with these observations. The whole of the cortical area from which the optic radiations are said to spring had been destroyed, with the exception of a small part of the parietal and temporal lobes, which have been shown in the normal brain to contribute fibres to the optic radiations as these approach close to the basal ganglia. Corresponding to this condition of the cortex, only a few fibres were found in the retrolenticular portion of the capsule, and these, with the exception of some peduncular fibres at its upper part, passed from the optic thalamus in the direction of the temporal and parietal remnants.

The area of cortex destroyed included all the convolutions in which the visual centre has been placed, and corresponding to this no fibres were found in the capsule passing to the external geniculate or anterior corpora quadrigemina.

Finally there were definite changes in the optic nerves and their central connections. — There was very marked degeneration of the external geniculate ganglion. Only a few cells were found in a small group in the anterior part of the nucleus, and the only fibres apparently connected with it passed between the outer surface of this group and the upper and outer part of the pulvinar. No fibres passed into the nucleus from the tract nor from the nucleus into the retrolenticular portion of the capsule.

The microscopical examination of the pulvinar gave no evidence of atrophy, yet macroscopically the posterior portion seemed to be deficient, in that it did not project backwards over the anterior corpora quadrigemina.

On macroscopic examination of the anterior corpora quadrigemina and their brachia there was no visible atrophy, and in the microscopical preparations no atrophy of cells was noted. In the Weigert-Pal preparations there was no definite evidence of deficiency of fibres in the anterior brachium nor in the anterior corpus quadrigeminum.

In the optic nerves and tracts there was a great deficiency of fibres, and such fibres as remained were of fine calibre and passed to the pulvinar and the anterior corpus quadrigeminum.

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[1] Vialet. *Loc. cit.*

The work of Monakow, Henschen, Vialet, Zacher and others, has shown that destruction of parts of the occipital lobes is followed by degeneration in the optic radiations, degeneration of cells and fibres in the external geniculate, loss of fibres and cells in the pulvinar, especially its outer and posterior part, degeneration in the anterior corpora quadrigemina, and in some cases in the optic tracts and nerves. In the anterior corpora quadrigemina Vialet found that the only alteration was a diminution in the number of fibres in the middle white layer.[1]

In the present case, in which it must be remembered the cortical lesion was one of very long standing, it is highly probable that there was some loss of fibres in the middle white zone of the anterior corpus quadrigeminum and in the anterior brachium, although from the Weigert-Pal preparations alone it was not possible to be certain of such a loss. The pulvinar also appeared, from the macroscopic examination, to be somewhat atrophied. But there was certainly no atrophy of the anterior corpus quadrigeminum nor of the pulvinar comparable to that of the external geniculate and optic nerve, and this was, in the case of the pulvinar, the more remarkable considering the almost total absence of the retrolenticular portion of the capsule.

Again, it was to these two nuclei which showed no marked atrophy—the anterior corpus quadrigeminum and the pulvinar—that the fibres which remained in the optic nerve passed, and the number of fibres having this distribution was apparently equal to that found in the normal brain.

Apparently, then, the only portion of the optic tract which had degenerated was that which passed to the external geniculate, and the degeneration of this part of the tract and of the external geniculate was the only marked result of the destruction of the cortex and of the loss of fibres in the optic radiations.

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[1] Vialet. *Loc. cit.*, *vide* also "Encyclopédie francaise d'ophthalmologie," Paris, 1903, chapter vii., p. 728, and *infra.*

Bernheimer has shown that in the monkey 70 per cent. of the fibres of the optic tract end in the external geniculate, whilst Monakow states that 80 per cent. so end in man.[1] Henschen has found, from clinical evidence, that the fibres which end in the external geniculate are alone connected with visual sensation. Pathological destruction of one or both pulvinars, or of the anterior corpus quadrigeminum or internal geniculate, does not interfere with visual sensation, provided the external geniculate be intact, but damage to the external geniculate causes hemianopia. From the external geniculate the path of visual sensation is continued to the occipital lobe by fibres passing in the lower part of the optic radiations.[2]

The present case agrees with these observations of Bernheimer, Monakow and Henschen, and shows that the fibres of the optic tract, which are connected with the visual cortex and, therefore, with visual sensation, form much the greater portion of the tract and have their only relay in the external geniculate.

Further, such fibres in the tract and nerve seem to be the fibres of large calibre.

Gudden's tract.—Where so large a part of the optic nerve and tract was destroyed, whilst the internal geniculate and posterior corpus quadrigeminum were intact, it was to be expected that if any tract of fibres existed in man, homologous to Gudden's commissural tract found in the rabbit, mole, &c., such a tract would be rendered conspicuous and would be easily traced. Such, however, was not the case, and there was no evidence of there being more fibres in the tracts than in the nerves.

This confirms Dejerine's statement, that no such tract has been demonstrated in man. In cases of bilateral atrophy of the optic nerves in man, Dejerine has always found Meynert's commissure, but has searched in vain for fibres homologous to Gudden's tract in the rabbit. Monakow, Henschen, &c., have, in their cases of bilateral optic atrophy, passed the question by in silence. Schlagenhaufer has, in such a case, described a commissure of Gudden, but from his figures this was undoubtedly Meynert's commissure.[1]

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[1] "Encyclopédie francaise d'ophthalmologie," p. 736.

[2] *Loc. cit.*, chapter viii., p. 749.

It is very much to be regretted that the eyes were not removed in the necropsy.

VII.—The Primary Cortical Lesion.

The changes in the thick cortex, corpus callosum, internal capsule and optic nerve connections were evidently secondary to the cortical destruction. The question now arises as to what was the cause of this cortical destruction?

The condition met with was not that usually described as "porencephaly," thus Monakow defines[2] porencephaly as "defects of the pallium in which either (1) the cortex is very thin and depressed inwards so that a collection of fluid exists between the arachnoid and the cortex, or (2) the cortex after being thinned out fails entirely, and the ventricle openly communicates by one or more openings with the subarachnoid space." In both these cases the cyst is described as lying between the arachnoid on the outer side, and the pia-mater covering the depressed cortex on the inner side, the pia in the second type being often continuous with the ependyma. The term, even with the above definition, embraces a number of pathological conditions, the commonest, perhaps, being the picture resulting from a cortical destruction due to arterial damage. The view has been put forward by Herchl, and denied by Monakow, that a porencephaly, such as is defined above, may be the termination of a fœtal hydrocephalus.

In the present case the condition met with was that of a general hydrocephalus in which the resulting thinning of the cortex was in a large and strictly bilateral area so severe as to form a thin cyst wall of neuroglia lined by ependyma and covered by pia-arachnoid.

That such a condition arose in very early life and in all probability intra-utero, is shown by the history of the case, by the accompanying microgyria and arrest in development of the calvarium and of the vessels supplying the cystic cortex, and by the conditions described above as secondary atrophies.

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[1] Dejerine. *Loc. cit.*, Tome II., pp. 432, 433.

[2] Monakow's article in "Lubarsch."

These secondary atrophies were obviously the results of a long-standing lesion, and the entire absence of sclerosis suggested that they were caused by an arrest in development rather than a degeneration.

Soon after birth the patient was certified to be totally blind, and there is no evidence in the life history that the cortical destruction was a progressive one. It is true that the patient walked in early youth, but latterly never moved without assistance; as however, no attempts were made to induce her to walk when she first showed a disinclination to do so, it is possible that this loss of spontaneous progression was the outcome of deficient will power, followed by prolonged disuse. The microscopical examination of the brain failed to give any evidence of recent degenerative changes.

The thickening and adhesion of the membranes about the foramen of Monro point to the hydrocephalus being the result of a pathological virus rather than a developmental error, and this view is borne out by the absence of developmental abnormalities in other parts of the body. What this virus was and why the destruction of cortex resulting from the hydrocephalus was so strictly limited and so bilateral can only be matters of conjecture.

A similar case of hydrocephalus accompanied by destructive thinning of the greater part of the cortical mantle is described by Dejerine and Long.[1]

Case of Dejerine and Long.

This case (Cas Longery) was that of a full-time male child, who lived from six to seven weeks, but was never seen to move any of its limbs. The only cerebral convolutions were limited to the infero-internal aspect of the hemispheres, and the area of convoluted cortex had everywhere a sharply-marked border defining it from the thin translucent cortex. Secondary to this defect in the cortex there was complete absence of the projection fibres, which constitute the cerebral peduncles. The authors do not discuss the possible ætiology of the lesion.

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[1] *Detailed description*, Dejerine, *Anatomie des Centres Nerveux*, Tome II., p. 187; *Resumés*, J. Dejerine and E. Long, *Société de Biologie*, 1898, p. 1181, and Edward Long, Thése de Paris. "Les voies centrales de la sensibilitié générale," 1899, p. 89.

VIII.—Physiology.

Finally, from the life history of the patient it is possible to estimate the physiological effects of the cortical destruction, and to see how far these agree with the present views on cortical localisation.[1]

The olfactory sense was present and played an unusually important part in the choice of food, replacing the lost sense of vision. That it was not interfered with by the lesion was to be expected, seeing that all those parts of the brain were intact which, from the study of comparative anatomy and such experimental and clinical evidence as has been obtained, have been associated with the sense of smell. Those parts are:—the olfactory bulb and tract, the uncus, the hippocampal convolution, hippocampus major, dentate convolution, nerves of Lancisi, peduncles of corpus callosum, fornix, fimbria, anterior commissure and the callosal convolution.

The sense of taste, the cerebral centre of which is probably in close association with the olfactory, was also present.

The neuromuscular mechanism.—The limbs could be moved at will and the sphincters were under voluntary control. There was, however, latterly weakness in muscular movements and inability to walk without support.

That the motor area of the cortex—applying the term to the area containing the cells which give origin to the pyramidal projection fibres—was not interfered with was shown by the perfect condition of the genu, and the posterior limb of the internal capsule and of the pyramidal tracts.

In this connection it is interesting to recall the condition of the ascending parietal convolutions. Not only were these absolutely and relatively extremely small, but on microscopic examination it was seen that, where the cyst had origin from this convolution, the adjacent portion of the convolution was entirely devoid of nerve cells.

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[1] Schäfer's article on Cerebral Localisation in "Schäfer's Text-Book of Physiology," vol. ii., 1900, where full references will be found to other authors.

That such a condition of the parietal convolutions was compatible with an anatomically perfect pyramidal tract, is consistent with the observations of Sherrington and Grunbaum, that in the anthropoid apes the postcentral convolutions are electrically inexcitable and their injury is not followed by degeneration in the pyramidal tracts.[1]

No marked wasting was seen in the limbs and the anterior horn cells of the cervical and lumbar enlargements of the cord were healthy. It is reasonable, therefore, to suppose that the talipes equinovarus noticed in the necropsy was the result of long sitting in the tailor position and not of a spastic paralysis.

It is difficult to understand why the patient, who had once been able to walk spontaneously and unaided, lost that faculty; had the loss been accompanied or preceded by a fit, or by a loss of muscular power, it would have clearly pointed to a progression of the destructive lesion, but no loss of power could be noticed at the time, and the behaviour of the patient was such as to make it appear that there was a disinclination rather than inability to walk. Her mother made no effort to overcome this disinclination, and it is possible that the power of spontaneous and unaided progression was ultimately lost through disuse.

Of muscular and tactile sense little can be said. That she could lift and play with small objects, although blind, and could modify the force of her muscular efforts, for instance using her hands for putting food into her mouth, or striking violently when angry, points to the integrity of these senses in the arms at least. The sense of touch, however, did not seem to play an important part in her appreciation of the external world.

As regards speech, the neuromuscular mechanism of articulation was normal.

The phonogenetic centre must have been present, and this must have been in connection with the auditory centre to enable her to learn to speak.

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[1] Sherrington and Grünbaum, *Proc. Path. Soc.*, liii., 1902.

Not only did she recognise the sounds of certain words, but she associated certain facts with them, and could even associate sounds heard with words; thus she would repeat, "Annie is going to have some fish" on hearing fish being fried, so that she may be said to have possessed an auditory word centre.

The connection of the auditory word centre and phonogenetic centre with the higher centres of intelligence and memory seems to have been present, in that she could answer certain questions correctly in the affirmative or negative, and in that she could remember verbal promises. In considering the possibilities of her brain in this connection it must be remembered that no attempts were made to educate her.

Her hearing was acute, and, with the exception of the part played by the senses of smell and taste, her auditory sense seems to have been the link between herself and the outer world.

It was by hearing and not touch that people were recognised; on hearing depended such powers of speech as she learned, and it was by hearing that she recognised what was going on around her, as was shown, for instance, by her screams of anger when the furniture was moved preparatory to washing the floor, a proceeding which was associated in her mind with being disturbed from her usual seat.

As the result of experiments on the monkey, Ferrier placed the auditory centre in the upper part of the superior temporal convolution. Munk, chiefly from experiments on dogs, ascribed the function to the whole temporal lobe, and Luciani, in the dog, to the whole of the temporal lobe and "probably, also, the cornu ammonis." Schäfer and Sänger Brown found total removal of both temporal lobes in the monkey to interfere in no way with hearing, but Schäfer,[1] from the results of electrical excitation, the time of myelination and from clinical and pathological evidence in man, concludes that "its focal point is to be found in the superior temporal gyrus, and especially the upper part of that gyrus, and the gyrus transversus anterior."

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[1] "Text Book," vol. ii., p. 762.

In the present case the function of auditory perception was not interfered with, although the whole of the temporal lobe was destroyed except the small strips on each side, which have been described above as representing, at most, the temporal pole with the uncus, the anterior or lower two-thirds of the superior temporal convolution with the adjacent temporal operculum, perhaps the tip of the middle and inferior, and possibly a strip of the occipito-temporal convolutions.

Vision.—There were no signs of appreciation of vision as was to be expected, seeing that the lesion embraced all those portions of cortex in which the visual area has been located.

As has already been said, the chief interest in regard to vision is to be found in the secondary anatomical changes, which support the view of Henschen, that those fibres alone of the optic tract which pass to the external geniculate are concerned in visual sensation. The functions of those fibres which remained apparently in normal numbers, and passed to the pulvinar and anterior corpus quadrigeminum, can only be conjectured, since the notes taken at Moorfields have been destroyed. They may have been afferent fibres for pupillary reflexes, perhaps also for other light reflexes, but the only evidence in favour of this view is the statement of the patient's sister, a sharp and observant girl, that nothing unusual was noticeable in the pupils, and the fact that no atrophy was found in the oculomotor nerves.

The inexcitable area.—The cortical visual area in man is thought to be of less extent than in the monkey, and it is practically certain that it is limited to the cortex of the cuneus and adjacent inner surface of the occipital lobe; according to Henschen, indeed, it is limited to the lips of the calcarine fissure. An injury, therefore, limited to the inner surface of the occipital lobe would have been sufficient to cause the loss of visual sense suffered by the patient.

What physiological loss is to be attributed to the destruction of the outer surface of the occipital lobes (with exception of the ascending parietal, the anterior portion of the supramarginal convolution and, on one side, a small piece of precuneus) and the greater portion of the temporal lobes? This area corresponds to the inexcitable area known as the parieto-occipito-temporal association centre of Flechsig.

Anatomically, the loss of this area caused no loss of projection fibres in the pyramidal tracts, though it doubtless accounted for many of the fibres lost from the retrolenticular portion of the capsule, for the atrophy of the posterior part of corpus callosum and for the deficiency in the fine radiating fibres in the frontal and parietal cortex.

Physiologically, we know that the mental condition of the patient was deficient and that her intellectual faculties were not developed, but the fact that she was blind from birth, and no attempts were made to educate her, might alone be sufficient to account for this, and considering how much she learned unaided it is not inconceivable that, had she as a child had careful tuition, the cortical destruction, great as it was, might have been not only compatible with life, but with the exercise of the higher intellectual faculties.

In conclusion, I should like to thank Dr. Redcliffe Salaman, in whose department at the London Hospital most of this work was done, for untiring help and advice, and Dr. Farquhar Buzzard for kindly looking through many of the microscopical preparations and for putting his laboratory at my disposal.

Reviews.

Des scléroses combinées de la moëlle. By Dr. O. CROUZON (Steinheil, Paris, 1904).

Dr. CROUZON introduces his subject with the following classification:—

(1).—Congenital and familial types:—(*a*) Friedreich's disease; (*b*) hereditary cerebellar ataxy; (*c*) familial spastic paraplegia.

(2).—Acquired:—(*a*) the tabetic form; (*b*) the form occurring in general paralysis; (*c*) the spastic form; (*d*) the arterio-sclerotic form of old people; (*e*) subacute combined sclerosis (associated with anæmias, cachexias and intoxications).

The work deals with the acquired types only.

The tabetic type of combined sclerosis is to be recognised clinically by the presence, in addition to the usual symptoms, of tabes, of (1) dragging of the feet in walking; (2) marked paraparesis; and (3) the extensor response in the plantar reflex.

The presence of painful cramp and of Strümpell's phenomenon are additional positive signs.

While this form of disease may be purely systemic it is more frequently non-systemic, and the author considers meningeal and lymphatic lesions to be important factors in its causation.

(Such a distinction between cases of which the clinical background is one of tabes dorsalis seems highly arbitrary.)

In general paralysis of the insane Dr. Crouzon finds that degeneration of the lateral and posterior columns is much more frequently present than the clinical pictures of such cases indicate.

Under the heading of spastic combined sclerosis are grouped:—

(1) Cases of spastic paraplegia in which the common signs of tabes co-exist, such as reflex iridoplegia, lightning pains, Rombergism and ataxy.

(2) Cases of amyotrophic lateral sclerosis in which degeneration of the posterior columns and cerebellar tracts exist. To these the term "parético-amyotrophic combined sclerosis" is applied.

It may be here remarked that in advanced cases of amyotrophic lateral sclerosis there is hardly a system in the central nervous organs which may not show conspicuous degeneration, but this fact does not render the inclusion of these cases as "combined sclerosis" in any way justifiable.

(3) The ataxic paraplegia of Gowers.

(4) An ataxo-cerebello-spastic type.

(5) Cases of primary spastic paraplegia (? cerebral diplegia) in which degeneration of the posterior columns is found *post mortem*, but is not clinically evidenced during life.

(6) Cases of syphilitic spastic paraplegia.

One cannot but regret the inclusion of groups 2, 5, and 6, even in a brief description. If the term "combined sclerosis" is to include all diseases in which degeneration of the lateral columns and of the posterior columns occurs, notwithstanding that they have nothing in common, neither clinically nor pathologically, then the term loses its value as signifying a definite clinical and pathological entity to which it has been very properly restricted by some authors, namely, the group "*e*" of Dr. Crouzon.

Moreover, the six groups by no means exhaust the cases where spastic paraplegia exists and where degeneration of the posterior and lateral columns is found, and no mention is made of the frequent occurrence of such degeneration in association with gross lesion of the brain and spinal cord.

That combined sclerosis occurring in old people with atheromatous vessels was first described by Demange in 1884 is surely an error. The condition was described many years previous to that date by the late Dr. Moxon, and the term "Moxon's paraplegia" was at one time in common use in this country.

Brief reference is made to the published cases in which the authors have considered that combined sclerosis has resulted from pernicious anæmia. It is pointed out that the clinical aspect of some of these cases has been widely different from that of subacute combined degeneration associated with severe anæmia.

The nervous derangements which occur in pellagra, in ergotism, in lathyrism and in cachectic states, are briefly alluded to. Dr. Crouzon does not consider their inclusion under the term "combined sclerosis" justifiable.

In ergotism a pseudo-tabes is the type most frequently met with, whereas in lathyrism and in pellagra a spastic paraplegia is the rule.

Subacute combined degeneration is described in great detail.

A topographic and pathogenic classification of combined sclerosis is tentatively put forward as follows :—

Topographic classification :—(*a*) Combined lesions of the posterior columns and of the lateral pyramidal tracts. This is the most common type. (*b*) In addition the direct cerebellar tracts are affected. Some of the tabetic forms fall into this group. (*c*) In addition the anterior columns are involved.

The author points out that these three groups do not correspond with clinical types.

Pathogenic Classification.

(*a*) *Systemic degeneration* :—

(1) Primary. The existence of such cases admits of doubt.

(2) Secondary. (*a*) Of exogenous origin and secondary to root lesions, &c. (found in tabes and general paralysis). (*b*) Of endogenous origin (found in general paralysis and in pellagra).

(*b*) *Non-systemic* :—

(1) Due to vascular lesions.

(2) Due to meningeal lesions.

(3) Due to lymphatic lesions and stasis.

The above classification is tentative only; the question of lymphatic lesions is suggestive and important.

An account of 56 personally-observed cases follows. These are most ably described and are accompanied by excellent clinical and pathological illustrations. A full bibliography is appended.

This thesis of 176 pages, with two plates and nineteen illustrations, has been prepared with great care, and the literature of the subjects is so fully dealt with that for this reason alone the book would be invaluable. Dr. Crouzon has written without bias. He has included everything that other authors have chosen to stamp with the term "combined sclerosis," and if conditions are described under this term without justification the fault lies with these writers rather than with their reviewer.

One cannot but regret the use of the term "combined sclerosis." Perhaps in none of the conditions included is the glial change the primary morbid change. Moreover, in the subacute forms is there ever any neuroglial increase?

As a history of the subject the work is complete. As a non-critical review it is excellent; the writer is content to place the work of his predecessors, most intelligently arranged, before his reader, but he leaves comment and criticism to the latter. He passes over certain works with brief reference, and this silent comment on their value argues that he has purposely avoided controversy.

The personal observations possess all the details which make them valuable for reference. The pathological considerations are suggestive, but do not go very far.

The classifications are not satisfactory; they seem to be determined by the literature of the subject.

JAMES COLLIER.

Die Migräne, von Dr. P. J. MÖBIUS. 2te Aufl. (Hölder, Wien, 1903).

THIS, the second edition of a well-known monograph, of which the first edition appeared ten years ago, is an excellent account of the clinical syndrome known as migraine. The conclusions at which Möbius arrives are summarised by himself as follows:—Migraine is generally a form of degeneration, hereditary in most cases, by direct inheritance from one or both parents. It is the product of morbid brain changes, hemicranial, whereby the patient has migrainous paroxysms from time to time, sometimes associated with various exciting causes, sometimes without. The form of attack is not always the same. The only feature common to the attacks is the occurrence of paræsthesiæ, cerebral in origin and entirely or preponderatingly unilateral. A complete attack consists of premonitory symptoms, aura, headache and vomiting. Incomplete attacks are more common and in them we find headache alone, or headache accompanied by nausea or vomiting. Sometimes the aura is all that occurs. Attacks may be superadded on one another, producing what he terms the "status hemicranicus" (an expression the latinity of which has a curious flavour). These various symptoms may also occur in other cerebral diseases apart from simple migraine.

Möbius discusses with clearness and detail the symptoms and course of the disease. There is an instructive and humorous chapter on treatment, in which he lays more stress on inter-paroxysmal hygienic measures and on the removal of possible sources of peripheral irritation than on treatment during the attack itself. Most of the drugs recommended, and their name is legion, yield best results if taken during the premonitory stage.

Once the paroxysm is on, the only reliable drug is morphia, which should only be given in selected cases.

The author frankly admits his inability to provide an adequate hypothesis which will account for the clinical phenomena. He contents himself with expressing his belief that the visual aura is referable to stimulation of the occipital cortex, that the headache is probably due to stimulation of the dura mater and that the vomiting is of cerebral and not of gastric origin. He dismisses the vaso-motor theory as inadequate, pointing out that in no case of disease of the cervical sympathetic, whether destructive or irritative, have symptoms of migraine appeared. Spitzer's theory is referred to in a foot-note, apparently while the volume was passing through the press. Möbius admits its ingenuity, but does not discuss it in full.

Not the smallest of the merits of this work are its clear diction and witty style.

PURVES STEWART.

Lectures on Diseases of the Nervous System. Second Series. By SIR WILLIAM R. GOWERS, M.D., F.R.C.P., F.R.S. (London, J. and A. Churchill, 1904.)

THESE ten lectures, which have already appeared in print at various times, are now, after having been revised, published in book form.

The first lecture deals with subjective visual sensations, and those occurring in epilepsy and migraine are considered in detail. The author concludes that there is very strong clinical evidence in favour of the existence of a higher visual centre in the region of the angular convolution, in which both fields of vision are represented, but that of the opposite side to a greater degree, and that without the assumption of such higher centres a large number of the facts of disease cannot otherwise be understood. There are also in this lecture some very interesting remarks on the relation between sight and movement, illustrated by clinical observations. Lecture 2 is upon the subjective sensations of sound, and in it will be found an interesting account of the nature and pathology of the various kinds of "noises in the head." It is followed by a note on the "Designation of Musical Notes in Science and

Medicine." The title of the third lecture is "Abiotrophy: Diseases from Defect of Life," and here Sir William Gowers takes the reader over the chief points in connection with premature death depending on defective vitality. Examples of abiotrophy are shown in the cutaneous, muscular and nervous structures, and in connection with the last-named the influence of interstitial growth is considered. Lastly, some special forms of degeneration of nervous tissues which seem to be abiotic in character are considered, as, for example, optic atrophy and Friedreich's disease. The next lecture (No. 4) also deals with myopathies of various kinds. Metallic poisoning from lead and arsenic forms the subject of the fifth lecture, and, among the useful lessons to be found here, that of the value of the lead line on the gums is especially emphasised in cases where the diagnosis is difficult. Nos. 6 and 7 deal with the effects of syphilis on the nervous system. The first of these deals with the general syphilitic processes, while the second, which is entitled "Inevitable Failure," is a study of syphilitic arterial disease and the effects of thrombosis, and the small amount of improvement that is likely to be found after an artery has once closed are considered. Syringal hæmorrhages into the spinal cord and the indications which suggest their presence form the subject of the eighth lecture, and the ninth is a valuable contribution to our knowledge of the disease known as myasthenia, and here the importance of the "nasal smile" and ophthalmoplegia in this affection is pointed out. The last lecture is on the "Use of Drugs." It will readily be gathered from these brief remarks that this series of lectures is one of great educational value, for Sir William Gowers has the art of showing how the most striking lessons can be learned from things that are seemingly commonplace and of little importance. But this is not all, for in addition to the presentation of that which is known in such a manner that makes it always fresh and interesting, there is in these lectures a rich collection of new observations and suggestions which throw a light on some of the most difficult problems in neurology.

H. Campbell Thomson.

Notes on the Composition of Scientific Papers. By T. Clifford Allbutt, M.A., M.D., LL.D., D.Sc., F.R.C.P., F.R.S., F.L.S., F.S.A., Regius Professor of Physic in the University of Cambridge, &c., &c. (London, Macmillan, 1904.)

Everyone who has ever written a paper on a scientific, or on any other, subject; everyone who is writing, or who contemplates writing, or who desires, or hopes, or aspires to write such a paper; and everyone who has read, reads, intends to read, may read, or may be about to read, any form of composition in the English language, ought to possess and master this excellent little volume. It will teach him, not only to detect bad writing, and to identify the qualities in which its badness consists; but also to appreciate good writing, and to recognise the qualities to which its goodness is due; and it ought to go far to prevent the perpetration by its readers, at least of the blunders, if not of the inelegancies also, which disfigure and deface so much of the writing on scientific and on other matters which is brought forth with such copious abundance.

Detailed notice or copious extracts are scarcely needed, for the reader, whether of scientific or of other matters, ought to read and assimilate every sentence in the book; and the writer must do so; but it is meet to call attention to certain features; and having done this, I shall presume to argue some minor matters on which I am at issue with Professor Allbutt, and to suggest certain additions which I think might be of use if made in a future edition.

The main thesis which the book contains, supports, and is written to enforce and drive home, cannot be too closely laid to heart. It is, that good writing means good thinking. If a writer does not express his meaning clearly, it is because he has not made it clear to himself. If his writing is obscure, shambling, confused, illogical, lacking in grip and precision, it is because it faithfully reflects the qualities of his thought. By their works ye shall know them. By a man's literary style you may gauge the quality of his intellect. In these days, when pretentious obscurity poses as profundity, and is widely accepted as profundity, this is a lesson that needs to be taught. It should be driven into the minds of men by frequent insistence; it should be proclaimed from the housetops. "Style and matter," says

Professor Allbutt, "can no more be dissociated than skin and bone; if we write clumsily, loosely, or disjointedly our thoughts are accordingly. In scientific prose words should be used as carefully as symbols in mathematics." Again "slovenly writing is slovenly thinking, and obscure writing for the most part, confused thinking." "Slovenly writing is not only for the most part slovenly thinking, but slovenly habits of expression corrode thought in its very substance." "By disorderly writing, then, we fall into worse things than muddle: we blunt the probity of our minds; we slur over difficulties and cover up ignorances. Content to be slovens, we lose our respect for truth, and blunt our consciences." If this is so, and no one who is at pains to write clearly can doubt it, then, conversely, to teach a man to write clearly will help him to think clearly also; and thus Professor Allbutt's book fulfils a double function. But let no man suppose that he can either think or write clearly by the mere resolution and effort to do so. "As strength and general dexterity do not suffice to make a cricketer, so knowledge and mental power do not suffice to make a writer. No one feels vexed that he cannot dance, paint, or ride to hounds without practice; yet men are apt to murmur that it is but the mere knack of writing, a knack with which, according to them and Dogberry, some fellows are endowed by nature, which is wanting to make their learning and talent conspicuous. To those who have taken lifelong thought how to write, who have striven painfully with the craft of this supreme art, the view of it as a happy gift seems a flippancy. . . . . . Let the candidate be assured that an easy and interesting style, like easy cricket, implies hard practice; the prose which in Swift, in Newman, in Froude, in Thackeray, runs so transparently that, to him whose eye is not set for it, the medium is unseen, comes of patience as enduring and training as exquisite as the more effulgent phrase of Sir Thomas Browne, Professor Ruskin, or De Quincey." He must serve a long and laborious apprenticeship who would become a master in this craft.

Professor Allbutt gives an interesting description of his own method of composition, which the student will do well to ponder, and to compare with Darwin's, as described in his "Life and Letters." Of methods there are probably as many as of men who use them, but each can learn something from the methods of others.

In this, as in all Professor Allbutt's writings, the general course of the argument, always clear and pleasant to read, always commanding attention, now and then sparkles with epigram; now and then rises to a fine eloquence. From two adjoining pages I cull the following:—

"Pedantry signifies not precision and nicety but a defective sense of relative values, and a stationary if acquisitive mind. Precision and nicety are not against simplicity but for it; they make for the shortest way and the least expenditure. Finicking and preciosity, on the other hand, are an otiose labour of saying common things in uncommon ways."

"Speaking generally, the greater the mere dexterity, the more obviously cunning the detail, the lower the art."

"An unchastened love of letters, welcome as it is in the young, is readily caught by uncommon words and phrases, as a girl is attracted by gauds; so it is in the essays of young authors we note violent metaphors, dashing phrases, archaisms, odd spellings, slang, split infinitives, and such tinsel; and the clever young men turn upon us with the exclamation—why should I not stick these gems, old or new, into my writing if I think them effective? Because, young man, you may not paint and patch your mother's cheeks. Dauber as you are, go down on your knees before your noble inheritance—the noblest speech that the world has seen: when you have known its loveliness, when your ears have opened to its melodies, when you have counted but a little of its riches, you will shudder at the thought of retouching a masterpiece."

Having commended the book, with all the urgency of which I am capable, to both the scientific and the general writer and reader, I will now discuss some matters, dealt with by Professor Allbutt, in which I venture, with diffidence, to disagree with him.

He deprecates definition. "As the departments of science become more and more complex, definition is recognised as a scholastical task which makes for sterility." While I should fully agree with him in warning the writer to distinguish between short descriptions and definitions, I can scarcely put too highly the value, as a mental discipline, and as an aid to clarity of thought, of working out definitions of the things we are writing about. I will never admit that anything can be separately contemplated by the mind, that cannot be specifically defined in words; and in many cases,—I speak from experience—it is only by the attempt to define, that we discover that what we thought was a separate thing is a mixture of things; or a part of some larger thing; or includes something that we thought was foreign to it; or is, perhaps, not a thing at all, but a figment.

In his warnings against faults of grammar, and his abundant collection of terrible examples, there are a few that he has overlooked, probably not having met with instances in the essays that it is his express purpose to criticise and improve. One is the use of the wrong auxiliary in verbs of motion, and in the imperative :—" He has (is) come." " They have (are) gone." " You have (are) to notice." Another is the degradation of the verb " get " into an auxiliary, and its frequent misuse in other ways : " He got (was) choked." " He got (reached) home." " You *have got* (!) to notice." The blunder was ridiculed sixty years ago by a master of English :—

" No matter who—no matter what condition, age, or sex,
But some 'get' shot, and some 'get' drowned, and some 'get' broken necks ;
Some 'get' run over by a coach ; —and one beyond the seas
'Got' scraped to death with oyster-shells among the Carribbees ! "

But I understand that Ingoldsby, with many other good writers of his time, is no longer read ; and, judging by the result, I imagine that writers of scientific papers take, as their model, the latter-day popular novelist.

" Beware of *the intrusive not*," says Professor Allbutt, " *e.g.* I ran to see if I could (not) get a seat," " Let us see if we can (not) help him." Here, it seems to me, the condemnation is too sweeping. I should aver that the introduction of the " not " into such sentences carries with it a new shade of meaning. It may very well be that, in the instances cited, there was no necessity or intention of introducing this modification, and in this case the word is " intrusive," redundant and misleading ; but, to my mind, " Let us see if we cannot help him " bears a different meaning from " Let us see if we can help him." The latter phrase implies that the speaker is quite uncertain whether help can be given or not. The former carries with it the opinion that the victim probably can be helped.

" Keep down your ' thats,' " is the author's excellent advice ; " *they multiply like lower organisms*."

Excellent, also, are his sections on the order of clauses, and on suspensions. In the latter he damns the split infinitive with a wealth of objurgation ; and in this he is, no doubt, in accordance with the best practice ; but in my own private opinion, which I put forward with delicacy against such a weight of authority, there is something to be said in favour even of this scouted and derided mode of speech. In as far as it does suspend the sense unpleasantly and irksomely, it is to be avoided ; but I do not see that it is in itself abominable, unless there is something in the infinitive mood that is on a level above or below the other moods of the verb. Let us thus qualify the moods and tenses:—

I, we, they, seldom use the split infinitive.

I, we, they, seldom have used the split infinitive.

I, we, they, seldom shall, may, can, might, could, would, should or ought to use the split infinitive.

I, we, they, seldom would have used, may be, or may have been, about to use the split infinitive.

Of, for, or by seldom using the split infinitive.

Use thou seldom the split infinitive!

Let him seldom use the split infinitive!

Seldom using the split infinitive.

If these are all good English, and who will deny that they are? then why is it wrong to say

To seldom use the split infinitive?

In the use of what Professor Allbutt calls "that flighty little word 'only,'" are many pitfalls, which he duly indicates for our avoidance, giving apt illustrations from published or unpublished instances, with their corrections. My own practice is not quite orthodox, in the sense of following that of standard English classics, and is therefore not the same as that of Professor Allbutt, who does follow them; but it has merits and conveniences which I venture to put forward. All would agree that the adjective "only" should immediately precede the noun that it qualifies:—"his only son," "my only hope," "the only reigning queen in Europe." Professor Allbutt, following the practice of most writers of repute and authority, usually places the adverb "only" also before the verb or phrase that it qualifies. This does not appear to me the best use. No one follows it on all occasions, and I think that the meaning is almost always clearer when the adverb succeeds, than when it precedes, the word that it qualifies. So long as the practice varies, it is difficult to tell whether the "only" is intended to qualify the preceding or the succeeding word. If it were always placed after the word that it is intended to qualify, there would be no doubt. To take one of Professor Allbutt's instances:—"He only touched the fringe of a large subject," which, says the author, signifies that the fringe was barely handled. I submit that it may mean this, or it may mean "he alone," or "only he," touched the fringe, &c. Professor Allbutt corrects the expression to "he touched only the fringe," but this seems to me open to a corresponding ambiguity. It may mean "he merely touched and did not handle," or it may mean "he touched the fringe and nothing else." Now put the adverb after the noun, and read "He touched the fringe only of a large subject." The ambiguity disappears. The sentence can have but one meaning. So, "I gave him only bread," may mean "I gave him bread and nothing else," or, "I gave bread to him and to no one else." But if we write "I gave him bread only," there can be no ambiguity. Be it objected that this is not a fair specimen, since the qualifying "only" is the last word in the sentence, and the difficulty still exists if we add a further clause; then I maintain that we prevent the ambiguity by proper punctuation. "I gave him bread only when he begged at my door" is ambiguous, it is true; but then it is unpunctuated. The sentence should be written "I gave him bread only, when he begged at my door."

And here I must lament that Professor Allbutt has not given more space to the exposition of the fine, but well-nigh lost, art of punctuation. I do not agree with him that commas are too frequent. They seem to me much too few. As compared with the great writers of the eighteenth and the early part of the nineteenth century, when the writing of English was most leisurely and most careful, and, upon the whole, at its best,—as compared with Gibbon, with Burke, with Johnson, with Hume, with Fielding, with Scott, with Coleridge, with Lamb, with Macaulay,—latter-day writers, in their use of commas, are parsimonious to the point of penuriousness; and the reader has to suffer unnecessary fatigue, in discovering for himself the relations to each other of the parts of the sentence, and in supplying the pauses, which ought to have been indicated to him by the writer. Let me compare two sentences of Professor Allbutt's own to illustrate the difference. "In progressive language and thought, then, a word, like a living creature, with which, being a function of life, it has much in common, undergoes, from its birth, a process of differentiation." In this sentence, every qualification, every suspension of the direct flow of thought, is indicated by a pause. Every time we come to a qualifying phrase, we are told to wait for the completion of the sense. We are prepared for what is to follow. We are warned that the meaning is not yet completed; and we suffer no fatigue. Compare this with another. "We do not always realise how lean and cursory a discourse may appear on the printed page which in conversation or public speech was telling enough." To this I will add what appear to me the appropriate commas. "We do not always realise how lean and cursory a discourse may appear, on the printed page, which, in conversation or public speech, was telling enough." In this punctuation, we are warned, by the pauses before and after "the printed page," to anticipate the antithetic clause "in conversation or public speech"; and the commas, which demarcate the latter clause, properly mark its interpolation between the pronoun and the verb. Again, "In scientific prose words should be used as carefully as symbols in mathematics." It appears to me that this most excellent counsel would be made even more impressive by the insertion of a comma. "In scientific prose, words should be used as carefully as symbols in mathematics." The addition of the pause,—the suspension of the utterance—lends emphasis to the phrase. Our expectation is heightened, our attention is set agog to learn what it is that the writer is about to tell us of scientific prose. The force of the sentence is increased; or so it seems to me.

Editors and printers are much to blame for the degradation of the art of punctuation. My request, a request which I am in the habit of placing at the head of my MSS., that my punctuation may be preserved, is not only frequently disregarded, but, when affixed to a letter sent to a leading journal, has even met with a polite but positive refusal. Now, I hold that it is as unjustifiable to tamper with the punctuation of a contributor, as to garble the words of his text. His meaning may be as much defaced by the one interference as by the other. All the more do I deplore that so high an authority as Professor Allbutt should join the congregation of ungodly and sinful men, who would diminish the importance, and impair the use, of the harmless, necessary comma.

On the choice of words, Professor Allbutt is very happy, "I note," he says, "that many of my candidates are content with one general noun—'question'; two adjectives—'marked' and 'localised'; one verb—'to involve'; one adverb—'markedly'; one conjunction—'while.'" We observe the same paucity in the vocabulary of many writers on scientific subjects. The strictures upon the misuse of certain words, particularly "theory," "type," and "fact," are loudly called for, and most valuable.

Pages 106 to 114 contain a sort of dictionary of terms that are commonly misused, which deserves very careful study. There is scarcely a word in the list whose misuse, in some scientific writing, has not aroused the ire of the critical reader; but one of them I shall undertake to defend, and one or two are absent whose misuse is not infrequent.

The word that seems to me defensible is "reliability." It is true that a certain odour of unorthodoxy clings about it. Professor Allbutt quotes the authority of the *Times* for the non-existence of the word; and it enjoys the distinction of being the solitary occasion of Mr. Gladstone owning himself convinced by an argument; so that he promised, it is said, that he would never use the word again. Nevertheless, I maintain that it is a useful word, and good English. The objection made to it is that it should be relyuponability, or nothing; that no such word as "reliable" can be constructed in accordance with the canons of English word-formation; that "reliability" is meaningless; and finally, that, if there were such a word, and if it did carry the meaning usually attributed to it, it would be superfluous; for we already have, in "trustworthy," a better word of equivalent meaning. None of these objections appears to me to have sufficient foundation. To object to the mode of formation of the word seems to place a pedantic and undue restriction upon the power of our language to produce new words. If we are forbidden to use "reliable" because it should, in strict logic, be relyuponable; so ought we to be forbidden to say laughable, or transferrable, or indispensable, to none of which exception is ever taken. Who would assert that such words are bad, and ought to be laughatable, transfertoable, or indispensewithable, respectively? But, it is said, such a word is not wanted; for we already have, in trustworthy, a better word which carries precisely the same meaning. This I should deny. We have Professor Allbutt's authority for stating that no two words are strictly synonymous; and I assert that the best practice is to use "trustworthy" of people, and "reliable" of things. Professor Allbutt is a trustworthy guide to English composition; the Westminster clock is a reliable time-keeper. In such use we find, I submit, a justification for the word. Professor Allbutt, in a private communication, begs that I will leave him the enjoyment of a "trusty" staff; and he has authority at his back. "Thou trusty pine, prop of my godlike steps, I lay thee by." But I contend that this use, dating from a time when there was no word "reliable," is obsolescent; and in this, I hope, my readers will agree.

A few omissions, in Professor Allbutt's list of misused words, come to mind as one goes through it. It would appear that the University of Cambridge, or, at any rate, its medical faculty, does not attract students from the west of St. George's Channel, or from the country north of the Tweed; for, if it did so, we should find the list enriched with a dissertation upon the respective uses of "shall" and "will," whose subtle distinctions are so strangely unappreciated by the acute natives of those remote regions. It is said, I know not on what authority, that, in the subeditorial departments of the great London newspapers, there are officials charged with the sole duty of correcting the "shalls" and "wills," the "woulds" and "shoulds," of those brilliant contributors, whose verbal communications are adorned with the ornament of a brogue. The tale seems unlikely on *à priori* grounds, since there is no other reason to suppose that the great London newspapers have any care for the purity of the language in which they are written; and equally unlikely *à posteriori*, since, in many of their cleverest articles, we can distinctly hear the brogue as we read, and the "shalls" and "wills" are distributed at random.

To set down all the extraordinarily subtle and nice distinctions which lurk in the uses of these little words, would be a task too onerous and lengthy for such a brief sketch as this; and, moreover, would be scarcely practicable in any case, for some of them depend upon inflections of the voice which cannot be reproduced by any written symbols hitherto devised. It is true that we can, by the use of italics, imperfectly mark the distinction between "Shall I *go?*" *i.e.* "Do you wish me to go?" and "*Shall* I go?" *i.e.* "Do you really wish me to go?" but we have no means of indicating in writing the distinction between "*Shall* I go?" with rising inflection, meaning "Do you really wish me to go?" and the same formula with falling inflection, meaning "Pray make up your mind whether I am to go or not!" and a third enunciation, in which the inflection on the words first rises and then falls, which implies "I shall not go, you will see if I do!"

These refinements of meaning we cannot indicate in writing, and although the broader and coarser distinctions can be so indicated, yet even they are so numerous that it is impracticable to enumerate them all. The differences between "shall" and "will" are, however, sufficiently wide to leave no excuse for disregarding them.

The word "shall" originally meant "owe." "I shall go," was "I owe" or "ought to go," and this derivation colours all the uses of the word, even when it does not remain pure and unaltered, as it does in "You should go," *i.e.*, "You ought to go." From obligation the transition is easy to

2. Command—Thou shalt do no murder.
3. Determination—He shall surely die.
4. Promise—If with all your hearts ye truly seek me; ye shall ever surely find me.
5. Inevitability—He shall perish everlastingly.

And so,

6. Simple futurity—Heaven and earth shall pass away.

But it is to be observed that, even in the last example, some flavour of inevitability still clings to the verb. The corresponding phrase, "Heaven and earth will pass away," is a much weaker prediction. "Shall" never is a simple prediction of futurity, unaccompanied by a strengthening certainty, except in conditional construction. "If he shall make default" contains no indication whether he is likely to make default or not.

Owing to this implication of inevitability, the phrase "I shall go" never means that the speaker is, at the time of speaking, exercising his volition. Either he is constrained by circumstances outside of himself to go, or he has at some previous time formed the determination to go. His going is not now a volitional act. For this latter meaning the use of "will" is reserved. "I will go" means "I now, this moment, make up my mind to go." "This is the choice that I now exercise." Nothing appears to a foreigner a more gratuitously perplexing vagary of English construction, than the replacement, in the second and third person, of the "will" of the first person by "shall"; and *vice versâ*. "I will, you shall, he shall." "I shall, you will, he will." But if we keep in mind the original meanings of the words, we shall see that the several constructions are by no means arbitrary. "You will," "he will," can never, it is manifest, be used in a sense strictly corresponding with "I will." The last expression proclaims a decision now formed in the mind of the speaker; but it is clear that he who says "you will," "he will," cannot communicate the corresponding information, for he has no means of knowing the changes in the minds of the persons of whom he speaks. These expressions are, therefore, set free for use in some differing but allied sense. They are used to express the sense of simple futurity, which resides, as we have seen, in a derivative meaning of "shall." "I shall go" may be used as a simple prediction of what is about to happen; and in such case the corresponding auxiliary in the second and third persons is "will." But "shall" divests itself with difficulty of its implication of inevitability or obligation; and "I shall" may, and often does, mean, "I shall be obliged to," as in "I shall die"; "I shall fall." When it is desired to express, in the second person, inevitability, obligation, or command, the auxiliary "shall" finds its proper use; and hence "If you look, you shall discover" means that you cannot help discovering—you will certainly discover; as compared with "If you look, you will discover," which signifies merely that it is most likely that you will discover. Similarly, "He will pay a fine of twenty shillings," leaves something, it may be much, to his willingness to pay; but "He shall pay a fine of twenty shillings" deprives him of all option in the matter.

The word "will," which primarily connotes volition, that is to say, choice exercised here and now, slides without difficulty into the secondary meanings,

1. Intention—Thy son, whom I will set upon thy throne in thy room.
2. Promise—I will never leave thee, nor forsake thee.
3. Undertaking—I will deliver it into thy hand this day.
4. Desire—He therefore that will be saved: must thus think of the Trinity.
5. Prediction—Sweet language will multiply friends: and a fairspeaking tongue will increase kind greetings.

The difference in meaning between "will" and "shall" is well illustrated, and is perhaps most pronounced, in the passive; and it is in this mood that mistakes are most often made. Yet it is sufficiently plain that "I shall be shot if I go" is very different from "I will be shot if I go." The former predicts what will inevitably happen if I go. The latter gives an undertaking or promise as to what I shall cause to happen if I go. In the former case I am a passive sufferer; in the latter, an active agent. Consequently, if we speak first of Tom and then of Dick, and say "He says he shall go," we mean "He (Dick) says he (Dick) is certainly about to go." But if we say, "He says he will go," we mean "He (Dick) says he (Tom) intends to go."

Keeping in mind the primitive meaning of "will," as exercise of volition, the absurdity of the interrogative "Will I go?" is apparent. It is asking my interlocutor to enlighten me as to the state of my own mind, which, of necessity, is known to no one but myself. It is permissible in one case, and in one only,—when I repeat, for the sake of emphasis, a question put to me. If one says to me "Will you go?" It is permissible to reply "Will I go? won't I!" as an emphatic colloquial assent.

From what has been said it is evident that the common Hibernicism "We would have to admit," is wrong. It is clear from the context that, under the circumstances, the admission is considered inevitable, and inevitability needs "shall" for its expression. What is meant is "We should have to admit."

This seems an appropriate place to mention another Hibernicism,—the failure in the *oratio obliqua*. "I asked him would he," for "I asked him if he would."

A very useful distinction of meaning can be expressed, in a conditional sentence, by a modification of the verb, which, nevertheless, is very rarely used. "If he is," carries with it the suggestion (as I say he is). "If he be," gives no indication of my opinion whether he is or not. "If he were," implies (as I say he is not).

To complete Professor's Allbutt's list of misused words would need a dictionary of considerable size, but there are a few instances, that have escaped his notice, instances so frequent and so vile, that it is hard to forego mention of them.

"Subjective" and "objective" are terms in frequent use by writers who appear to have but the dimmest glimmer of the meanings that should be attached to them. They are better avoided altogether.

"Class of state," "kind of case," are traps that few escape. We meet frequently with "this kind of case," and even "these classes of case," as if one thing could be divided into kinds or classes! "We administer strychnine to this class of case *where* the symptoms are severe." Sometimes the writer avoids one blunder, to fall into a worse—"These sort of cases."

The use of an adverb for an adjective is not rare by would-be superfine writers. "It smells badly." That is, it exercises the function of smelling, and performs this function inefficiently! All that he meant to say was, "It smells bad."

Professor Allbutt warns us that even the definite article is not to be used without discrimination. He might have extended the warning to the indefinite article, which is very frequently used improperly. Modern writers, almost without exception, disregard the proper use of the indefinite article before an aspirate. We read, in books by even the best writers, of "a hereditary tendency," "an humble meal," "a hotel," "an hospital," and so forth. The rule is, that when a word beginning with the aspirate has its accent on the first syllable, it is preceded by "a," as "a house," "a helpmeet," "a Huguenot," "a hospitable man." But when the accent is on any syllable subsequent to the first, the article to be used is "an." "An hotel," "an hydatid," "an horographer," "an hallucination." "An humble meal," "an hospital," are both wrong. Both come down to us from times at which they were right, but they are now become wrong because the pronunciation of both words is altered, though in different ways. Two generations ago, the aspirate in "humble" was silent; and "an humble ——" was consequently correct: as "an hour," "an heir" are to this day. But the frequent use of the word by Uriah Heep produced a violent repulsion to it in that form; and the fact, that Charles Dickens substituted an apostrophe for the aspirate, induced a host of ignorant people to suppose that the correct use was to sound the aspirate; so that the pronunciation of the word was altered, and, the aspirate being no longer silent, the proper form of article to precede the word is no longer "an," but "a." "An hospital" also is a survival, but from a different use. Many people, who desire to be punctiliously correct, are misled by the form of the article, and suppress the aspirate, believing "an 'ospital" to be the correct pronunciation. It is not so, however. The "an" comes down to us from a time when the accent was on the second syllable, and "an hospital" was correct. Now that the accent has been transferred to the first syllable, the article should be altered in correspondence, and "a hóspital" is the right form at the present day.

"Above" and "over" for "more than"—"above a hundred," "over a thousand,"—are uses that deserve the pillory; and the use of "above" as an adjective deserves both pillory and stocks. "The above statement," "the above paragraph," "the above figures," set one's teeth on edge. I suppose that, if there is an above statement, there may be an abover statement, and an abovest beyond that!

But the subject is endless; and, fascinating as it is, and powerfully as it lures us on, I must be content to finish as I began, by commending Professor Allbutt's book to the earnest attention of every writer and reader of English.

CHAS. MERCIER.

Why the Mind has a Body, by C. A. STRONG, Professor of Psychology in Columbia University. (New York. The Macmillan Co. 1903. pp. 355. Demy 8vo.).

PROFESSOR STRONG chose his title, he tells us, in order to put on record the pretension of panpsychism to explain the connection of mind with body. I think the title unfortunate, for it has a somewhat catch-penny air, that arouses in the reader an unrecognised prejudice, which is wholly unjustifiable, against the book. In fact, the title is the only part of the book with which fault can justly be found. In all other respects, the book is admirable. It is closely reasoned; it is thought out clearly and deeply; it is admirably lucid in exposition; it is perfectly fair; and, considering that the author is persuaded that he has finally solved the excruciating problem of the connection between body and mind, it is written with a modesty which is both creditable and engaging. Anxious as I am to find a solution, as a haven of rest after much weary speculation, I cannot, for my own part, feel full assurance that Professor Strong has reached the much desired goal; but he has certainly thought the matter out more clearly and to a farther extent than any of his predecessors; and appears to have reached a limit beyond which it seems improbable that we shall go, until some new intellectual calculus is invented, or some new faculty is added to human capacity.

Up to the present time, two hypotheses hold the field between them :—interactionism, which regards the brain as an instrument employed by the mind in its dealing with the world of objects; and automatism, which regards the brain as the physical basis or condition of consciousness, which goes on simultaneously upon another plane, accompanying the brain process without exerting any influence upon it. The latter is the faith held by most neurologists, and it includes, in fact, two widely different hypotheses. One of these is the "conscious automaton" hypothesis of Huxley, according to which consciousness is an *effect* of brain action, an effect which does not in its turn become a cause, and react upon the brain. There is a causal relation between body and mind, but the causation acts in one direction only. Mind has no causal efficiency on brain, though brain process is the cause of mind. The second hypothesis is the "parallelism" of Clifford, which is the "pre-established harmony" of Leibnitz restated. According to this, consciousness and brain action flow along side by side, the brain being no more responsible for the changes of consciousness, than consciousness for what happens in the brain. There is no causal connection, either in the one direction or the other.

The general importance of the choice that is made among these hypotheses is well indicated by Professor Strong. It lies at the bottom of the principle of conservation of energy; since on one hypothesis this doctrine is sustainable, on another it is not. It has a direct bearing on biology; for it opens the question of the part that consciousness takes in determining survival. It is, of course, intimately concerned with brain physiology, and with the order and disorder of mind. It lies at the root of philosophy, and affects enormously our conception of the universe. Finally, it must, though Professor Strong omits to point this out, have a profound influence on theology.

The book is divided into three parts; of which the first treats of the known facts that bear upon the problem. It treats in turn of the apparent action of mind on body, of the apparent action of body on mind; and of the apparent dependence of mind on body. It then considers, with great acuteness, the correlate of consciousness, and, after setting forth the several hypotheses with perfect fairness and clearness, it gives, with equal justice and force, the arguments for and against each, in so far as they depend upon the facts that already have been enumerated; and shows, that, upon these facts alone, no decision can be reached.

In order to complete the inquiry, it is necessary to discuss the metaphysics of the subject. Ere we can determine the relation of mind to matter, we should have some clear notion of what is meant by mind, and what is meant by matter. Has matter any existence, independently of mind? Is it purely phenomenal, or is there behind phenomena a *noumenon*? And mind—is this, too, made up solely of states of consciousness, which again are but phenomena, or does it offer a reality to our grasp? If there be a reality, is it in the train of conscious states, or is it in the subject to which these states are conscious? Here Professor Strong is very logical, but not very convincing, and here even his powers of exposition do not always succeed in making his meaning quite clear. In his final conclusions he rejects the distinction between subject and object-consciousness as an absolute distinction, and holds that "the Ego is a fresh experience as it comes, before we have had time to turn round upon it cognitively, and while we—that is, it—are engaged in cognising other things." The hypothesis is ingenious in the extreme, but upon close and careful examination it fails to convince. The whole section is a most subtle and profound piece of reasoning, and deserves the closest attention, but, when the author reduces his opponent's view to an absurdity, it is difficult to refrain from a *tu quoque*. "If there be a soul or subject, it is as impossible for me to be immediately conscious of my own existence as of that of other people. Another man's mind is not more inaccessible to me than the inmost essence of my own. I am cut off from myself; an impenetrable veil separates *me* from *me*. That our actual self should not be given, that we should be denied true self-consciousness and our essence made to consist in something other than thought and feeling—this, to speak frankly, is so great an absurdity that I hardly know a greater of which a philosopher can be guilty." This is no doubt extremely acute, but is not Professor Strong's own hypothesis open to a similar retort? "The Ego is the fresh experience as it comes, before we have had time to turn round upon it cognitively," but who is the *we* who thus turn round? Professor Strong sees the difficulty, and instantly confronts, or appears to confront it, for he goes on:—"and while we—that is, it—are engaged in cognising other things." It is very difficult for a psychologist, accustomed to current notions, to identify the ego with experience; but this, no doubt, is just what Professor Strong considers our weakness. Experience, in whatever sense the term is used, has always, until now, implied objectivity. But let us wrestle with the notion, and try to posit experience—an experience—as identical with the subject. What happens? This experience (which is I) is first engaged in cognising other things, and then turns round upon itself cognitively, that is, I suppose, contemplates or cognises itself. It may be incurable habit or [illegible] density, but for my own part I am unable to conceive an experience which is not *my* experience or someone else's experience. Experience I take to be that which happens; and I am unable to conceive an experience which does not happen to someone. In other words, the duality of subject and object in experience is to me inescapable, nor is the difficulty of escape lessened by my being bidden to imagine an experience cognising itself. First the experience cognises other things, and is Subject. That which cognises is what we call Subject. Then it turns round and cognises itself. Now it is become Object: that which is cognised we call Object. But, in becoming Object, it has not ceased to be Subject, so that it, this very same "experience," is at the same time both Subject and Object, both that which knows and that which is known. The feat of the Irishman who turned his back upon himself was a trifle in comparison. In a footnote Professor Strong explains that what, in the passage above quoted, he calls "experience," and distinguishes from knowledge, he later, by a usage which he believes professes a profound truth, calls immediate knowledge, as distinguished from representative knowledge. The explanation makes the matter no clearer to me. I am unable to picture to myself "immediate knowledge" cognising other things or turning round to contemplate itself. The only thing that I can conceive as knowing is *I*—that [illegible] spring of power which constitutes my Self.

If Professor Strong's meaning is, that that which was lately

Self can, by an artifice of memory, be contemplated by that which is now Self. Then there is nothing new in his position, which is only that commonly held. But this, I take it, is the position that he speaks of as so great an absurdity.

Next follows a discussion of things-in-themselves, by which Professor Strong understands "realities external to consciousness of which our perceptions are the symbols." This discussion is manifestly relevant to the issue, for, as he says, the relation of mind to body will evidently be an essentially different thing, according as the body is the symbol of a reality external to consciousness or only a phenomenon within consciousness. He first deals with what he calls "thoroughgoing phenomenalism," by which he means "the total and consistent denial of the possibility of transcending experience and cognising realities beyond." He shows without difficulty that this means solipsism,—that such a thoroughgoing phenomenalism makes no provision for knowledge of the minds of other men and of animals. They do not exist in the world of solipsism. Yet we are perfectly certain that they exist. We have therefore, unquestionably, a kind of knowledge transcending experience; a kind founded neither on reason nor experience but solely on "instinct." For my own part, while admitting to the full Professor Strong's argument against solipsism, founded on our certainty of the existence of other minds, I am unable to see that this certainty exceeds in kind or degree our certainty of the existence of matter. As we infer that, behind certain phenomenal experiences, there is a reality which we call minds of others; so, and not otherwise, do we infer that, behind other phenomenal experiences, there is a reality which we call matter. "The belief is there," says Professor Strong of the belief in other minds, "unshakable, in the minds of philosophers and laity alike." True, and so is the belief in matter. Whatever is true of the one belief is true of the other. Philosophers may, indeed, deny in words the existence of matter; but they cannot, by doing so, get rid of the belief. They may deny in words that resistance is inseparable from extension; but cannot, by so doing, get rid of the belief that it is inseparable. And this similarity of the two *beliefs* remains, even although the things believed in may be conceived as wholly different;—although, as Professor Strong acutely distinguishes, the reality we ascribe to matter is in its being a possible experience to ourselves, while the reality we ascribe to other minds is in their being actual experiences to *themselves*. Herein I do not so much differ from Professor Strong, as think that he might have taken up more ground than he has taken.

Then follows an extremely ingenious discussion of causality, in connection with the Kantian argument, forbidding the use of the causal category for the purpose of transcending experience. Taking account, as Kant fails to do, not only of phenomenal causality, but of what Professor Strong calls inter-mental causality, by which he means the causation that connects mental states, whether in the same or in different minds, he shows, and shows, I think, satisfactorily, that the Kantian veto does not hold good. And he goes on to argue with great cogency that, in order to fill in the gaps between individual minds, and give coherence and intelligibility to our conception of the universe, things-in-themselves must be assumed. Further arguments to support the same assumption he finds in the facts of physiology and in those of evolution; and finally, while admitting that things-in-themselves cannot be logically demonstrated, in which I think he admits too much, since his own demonstration is eminently logical, he finds that we are nevertheless driven to accept, and compelled to suppose, their existence.

Having established the existence of noumena, Professor Strong goes on to discover their nature, for he holds that, in conceding the impossibility of such knowledge, Kant and Spencer conceded more than they need have done. Some avenues of knowledge, he points out, are open to us. Since noumena manifest themselves through perceptions, these may throw light on the matter; and, pursuing this line of investigation, he concludes that it raises a doubt whether noumena are corporeal, or whether they are not mental. He then searches the source of our concept of reality, and is driven further towards the assumption of their mental nature. The analogy of other minds, and the evolution, both phylogenetic and ontogenetic, of mind out of noumena, complete for him the demonstration, and lead him irrevocably to the conclusion that things-in-themselves are mental facts.

He then turns back, and re-examines the three hypotheses of the connection of mind with body in the light of the conclusion that he has reached. He here analyses these creeds, as set forth by their chief supporters, and shows that each of the three is subdivisible into several minor varieties, clearly, if not widely, distinguishable. All of these, save one, he rejects in turn, and finally he finds rest for the sole of his foot in a theory of psychophysical idealism. According to this doctrine, the brain process, as we conceive it, is phenomenal. The dance of its molecules is a conceptual picture symbolising a reality, a thing-in-itself, a noumenon, which lies behind. Now, the only realities are mental. Things-in-themselves are mental facts; and the thing-in-itself which the dance of my brain molecules symbolises, the reality which lies behind this phenomenon is—my consciousness! Thus, current psycho-physical notions are turned topsy-turvy. Instead of an anatomico-physiological arrangement of energising cells and fibres, which forms the physical basis of mind; mind is the reality which underlies the phenomenon of cells and fibres. The concept takes away our breath!

The concept is not destitute of its own proper and inherent difficulties, as Professor Strong freely admits; and for the abolition of these difficulties he promises us another volume, to which we shall look forward with interested expectation. It is, however, a very daring speculation; it is worked out with a really beautiful dialectic, which is as fair and unsophisticated as argument can be; and if his main conclusion does not at once command our adhesion, he does, in the course of his long and continuous chain of reasoning, settle to our satisfaction many minor matters that have long been in dispute. While we cannot at present give an unconditional assent to his main thesis, we are happy in being able to congratulate Professor Strong upon having produced the most exhaustive, the most lucid, and perhaps the most persuasive treatise that has yet been written upon the subject.

Chas. Mercier.

Nervous and Mental Diseases. By Archibald Church, M.D., Chicago, and Frederick Peterson, M.D., New York. Fourth Edition. (W. B. Saunders & Co., Philadelphia, 1903.)

The fourth edition of this work has given the authors the opportunity of interpolating new matter of considerable importance. The authors state in their preface that the subjects which have received additional notice in the present edition are the healing of nerves, intermittent limping, herpes zoster and that form of epilepsy which is accompanied by myoclonus. Under the heading "intermittent limping" is described a condition otherwise known as "claudication intermittente," or angiosclerotic paroxysmal myasthenia. The condition is manifested by pain and paræsthesia of the feet and legs, which is intensified on attempting to walk. The feet are more comfortable in the dependent position. This condition of recurrent lameness has long been known to occur in the horse, and has been found to be due to interference with the circulation of the limbs. In man it is always associated with inefficient circulation in the legs, and in many instances no pulse can be felt in the dorsal artery of the foot.

The association of epilepsy with myoclonus is described as myoclonus epilepsy. It is said that the myoclonus, when associated with epilepsy, is somewhat atypical when compared with the essential myoclonias, and that the contractions may be both lightning-like, and also may have a fibrillary character. It is not obvious, from the description, how such contractions differ from those seen in myoclonus if the word is used with its ordinary significance. Under the title "Neuroses," a very large number of conditions are described, which manifest a disturbance of functional control, but are not marked by any known change in the neural apparatus. The authors state their belief that many of these affections will eventually be histologically classified—many of them would have been better so grouped, for much does not seem to be gained by placing in one group, under the heading "Trophoneuroses," such varying conditions as acromegaly, myxœdema, exophthalmic goitre, and Raynaud's disease. The work is well illustrated, and the articles contain the essential features in each disease. Some few inaccuracies seem to have crept into this work, and an instance of such is the statement that "Fraser reported a series of cases of family ataxy extending through several generations." Fraser reported a defect in the cerebellum occurring in a brother and sister, the parents and grandparents were perfectly healthy.

In the earlier portion of the work the authors insist on the importance of systematic investigation in nervous disease. The method for obtaining the knee-jerk, of which an illustration is given, is one which does not especially commend itself. A lateral blow does not, in a difficult case, elicit the knee-jerk with the certainty which results from the fall of a weighted hammer on to the patella tendon. Again, the method advocated for obtaining ankle clonus is one which may give rise to error—for a spurious form of ankle clonus can often be obtained with the leg in the extended position, which disappears when the leg is flexed.

The work is well illustrated, the figures representing the diseases pseudo-hypertrophic paralysis, tabes and paralysis agitans being especially good. The description of each disease is short and clear, and the whole forms an excellent text-book on nervous diseases for students. Dr. Church is responsible for the first part of the book.

The second part, on Mental Diseases, is the work of Dr. Peterson. In this edition the principal addition is a section by Dr. Adolph Meyer, of New York, consisting of a critical review of the German schools which have recently made such important changes in our point of view of psychiatry, and chiefly of the works of Ziehen, Kraepelin, and Wernicke. This section is well worth the attention of those who wish to gain shortly an insight into the views of German writers, without reading the originals. In the general etiology are a number of good illustrations of cranial anomalies, and the section on treatment is brought well up to date. Mental diseases are described under the following headings: mania, melancholia, circular insanity, epileptic insanity, dementia (secondary, senile, and primary), paralytic dementia, paranoia and idiocy, no new classification being introduced. Acute delirium is not described apart from mania, confusional insanity is not specially described, and katatonia is considered to be really a form of melancholia. There is no heading for stupor, and dementia præcox is not mentioned in the classification, although referred to in Dr. Meyer's section. Under paralytic dementia Dr. Peterson refers to Mott's observations as to the frequency of syphilis as an etiological factor. The pathological anatomy is dismissed in a short dogmatic list of gross and microscopical changes. Paranoia is defined as a progressive psychosis founded on an hereditary basis, characterised by an early hypochondriacal stage followed by a stage of systematisation of delusions of persecution which are later transformed into systematised delusions of grandeur. Reference is made to the views of Ziehen and Krafft-Ebing, but the question as to whether it should be considered a primary intellectual disorder in contradistinction to the so-called affective psychoses, which has occupied Continental writers so largely, is not referred to. The greater part of the chapter is taken up by the description of one case.

The chapter on idiocy is well illustrated by photographs of different types, and Preyer's conspectus of the development of the faculties in normal children, during the first forty months of life, is given, and forms a useful standard of comparison with the mental development of idiots.

F. E. Batten.
R. Percy Smith.

Ophthalmological Anatomy, with some Illustrated Cases. By J. HERBERT FISHER, M.B., B.S.Lond., F.R.C.S.Eng. (Hodder and Stoughton. 1904).

THE expressed object of this volume is to provide for the student of clinical ophthalmology a brief and concise description of the anatomy of the visual apparatus.

The author does not confine himself to pure anatomy, but deals with the physiological relations and functions of the structures he is describing, and with the symptomatology occasioned by their involvement in disease. An account of the anatomy of the eye-ball is purposely omitted. Nearly one-half of the book is devoted to the description of cases illustrating the clinical phenomena appearing in disease of the nervous structures subserving vision, in orbital cellulitis, and in thrombosis of the cavernous sinus.

Except as regards its brevity, the book is disappointing. The departure from the classic language usual in anatomical description does not, perhaps, add clearness nor emphasis. Exception may be taken to several unqualified statements, of which some are unduly at variance with generally accepted opinion, and others are upon matters so controversial as to lead the reader into error.

While dealing with the visual pathways and centres, the author states, "In many mammals, and in all vertebrates below mammals, complete decussation of the optic nerves takes place." No reference is made to the association of decussation with binocular vision and with parallelism of the visual axes. Since, in the opinion of the highest authorities, some degree of decussation is present in all mammals, and probably exists in some birds, this statement seems to require qualification.

In his account of the functions of the sixth nucleus, the author introduces a controversy which is of doubtful practical value. The reader is left to decide for himself between two theories, and it is not stated that one of these—that the sixth nucleus subserves lateral conjugate movements—is generally accepted, and that the clinical evidence in its favour is overwhelming. The statement that "fibres are added to the facial nerve from the third nerve nucleus, and these appear to be the fibres which the facial distributes to the frontalis orbicularis palpebrarum and corrugator supercillii; they descend from the oculo-motor nucleus in the posterior longitudinal bundle and join the issuing portion of the seventh root," argues that such fibres have actually been traced. Whereas, the existence of a synaptic tract has yet to be proved, and direct fibres certainly do not exist. That "the posterior longitudinal bundle is derived from fibres of the anterior pyramidal tract" does not convey to the reader the author's simple meaning, that this tract passes into the ventral column of the spinal cord.

The view that the capsule of Tenon forms a socket in which the globe of the eye moves is rejected, and the author's argument, that both the globe and the capsule move harmoniously together upon the retro-ocular tissues, has much in its favour. He insists upon the combined action of all the extra-ocular muscles in even the simplest ocular movement.

The descriptions of illustrative cases are excellent.

James Collier.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

AT the Clinical Meeting held at 11, Chandos Street on Thursday, April 21, at 8.30 p.m., the following cases were shown:—

Dr. JAMES TAYLOR.—*Cases of Tremor.*

(1) C. H., brass finisher, aged 62. Nothing significant in his family history or previous health. Has been troubled with tremor for twelve years. In the last six years it has been so bad that he has occasionally had to take stimulants to steady himself. Unable to work since August on account of tremor.

Tremor is of both hands, a little worse in right, is definitely increased on voluntary movement and becomes much more rapid on gripping strongly.

It is desired to elicit opinions as to the nature of the tremor—whether it is metallic in origin.

(2 and 3) Two brothers, aged 20 and 19, who have suffered from tremor since the age of 7. Up to that age each had been subject to fits. The fits ceased when the tremors commenced. Two other males and one female in the family are healthy. There is no knowledge of any similar complaint in the family. The two cases are curiously similar. The tremor is absent during sleep and complete rest. It affects both upper limbs, and is increased in movement and under excitement. The face muscles are also affected and so are the articulatory muscles, giving to the articulation a spasmodic and explosive character. There is no nystagmus, no increase or other change in the reflexes. The psychical condition is somewhat defective.

Dr. LEONARD GUTHRIE and Dr. RAYNER BATTEN.—*Intention tremors, most marked on right ride, associated with paresis of left superior rectus muscle, dilatation and inactivity to light of left* *pupil, unsteadiness of gait, marked "Romberg's sign," and active knee jerks.*

Bertha W., aged 30, single. In August, 1900, she had a fit in the street, was unconscious for twelve hours. Never fits before or since. The fit was followed by severe headaches, vomiting and giddiness, mostly on rising in the morning. She was in an infirmary for nine months, and was supposed to be suffering from intracranial tumour. Headaches and vomiting ceased on discharge from infirmary, and her general condition has remained unchanged for the past two years. Dr. Rayner Batten reported (April, 1902)—" Left pupil inactive to light, but reacting to accommodation, and greater than right pupil, paresis of left superior rectus. Fundus appears normal." She had, also, blunting of tactile sensation over left trigeminal area, scarring and perforations of soft palate. She gave history of bad sore throat and rash (attributed to eating too much salt) one year before onset of symptoms. Hearing has never been impaired. At times she has exhibited a persistent spontaneous jaw clonus. Ankle clonus and plantar reflex have been absent.

The condition points to lesions in the neighbourhood of the corpora quadrigemina, probably specific arteritis and thrombosis of branches of the basilar artery, rather than tumour. It will be noted that the sphincter, constrictor and superior rectus centres seem to be affected, whilst the levator palpebræ, which comes between the two former in the centres below the corpora quadrigemina, escapes. This suggests multiple lesions rather than a single one.

Dr. HEAD and Mr. ERIC GARDNER.—*Tremor in a case of family spastic paralysis.*

Rose A., aged 17, a Russian Jewess, the eldest of a family of six. She has been affected with gradually progressive difficulty in walking since early childhood. Her mother noticed tremor and nystagmus at an early age, certainly as early as three years old. At times her speech seems to be unaffected, at others she speaks slowly and hesitatingly and pronounces all her words very deliberately. She herself believes that she does not speak so well as she did.

Her gait is of a typical spastic type. She walks with stiff legs and swings them forwards from the thighs, inclining the body to the opposite side to enable the toes to clear the ground, but, nevertheless, the feet catch at every step and her boots are worn through at the toes and over the ball of the foot. She is able to walk quite rapidly, taking little short steps, and she is in no way unsteady. She never feels giddy and she has never fallen. When she wishes to turn quickly she stands on one leg and twists her body rapidly into the required position. There is no wasting of any group of muscles in the legs and muscular power is good, but there is considerable spasticity, which is more marked on the left side.

Her arms show no sign of wasting and the grasps are good. There is no rigidity, and no difficulty in relaxing the muscles after a voluntary movement had been made.

Her hands are affected with a very marked tremor on voluntary movement, which is more marked on the right side. The hands as they lie in her lap are kept perfectly still, but when an attempt is made to move them they are seized with a pronounced tremor. Her writing is markedly affected. She is unable to thread a needle unless it has a very large eye, and she is almost unable to pick up a pin; in attempting to do so she brings her hand rapidly to where it is lying and then fumbles at it till she picks it up, the tremor increasing the nearer the hand gets to the pin. There are no swaying movements of the body and head when at rest.

There is no incoördination. She is able to walk along a straight line without deviating from it. She is unsteady when she stands with her feet together, but this is accounted for by the deformity of her feet. She does not tend to fall with her eyes closed, though her unsteadiness is, perhaps, a little increased.

She is able to touch any part of one leg with the toes of the other foot. She can bring her two index fingers together accurately, and touch the tip of her nose with the eyes open or closed, but these movements are associated with considerable tremor, which makes them rather difficult of performance. There is no alteration of the sense of passive position. There is no alteration of sensation to touch, pain, heat or cold in any part of the body.

The superficial reflexes are all present. The plantar is present and is of a typical extensor type. The knee jerks are equal on the two sides, but are very exaggerated, a thigh clonus being obtained on tapping the patellar tendon. Ankle clonus is well marked, and the elbow and wrist jerks are very brisk. The jaw jerk can be obtained but is not exaggerated.

Vision has always been defective. She is able to see $\frac{6}{18}$ with the left eye and $\frac{6}{60}$ with the right. She has hypermetropia, and when this is corrected she is able to see $\frac{6}{18}$ with the left eye and $\frac{6}{36}$ with the right. The discs and fundi are normal in both eyes. Smell, taste, and hearing are normal.

There is no ptosis, no diplopia, and no paresis of any ocular muscles. Her eyes are in a state of constant fine horizontal movement, which is more marked if she fixes or turns her eyes to the extreme right or left. The movements of the right eye are much more marked than those of the left. The pupils are equal and react to light and accommodation. There is no facial paralysis, the palate and larynx are normal, the tongue is protruded in a straight line and is tremulous. The sphincters are not affected in any way.

There is a slight lateral curvature of the spine in the dorsal region, with the convexity to the left. The feet are in a position of pes cavus, which is more advanced on the left side. The mother of this patient, aged 38 years, has been affected with a tremor of the hands on voluntary movement for about six or seven years. This tremor is not so violent as her daughter's, but in other ways resembles it in all respects. She also shows a fine horizontal nystagmus when she looks to the extreme right or left. Her knee jerks are exaggerated, but the plantar reflexes have never been obtained. No other sign of disease can be found.

Besides the patient Rose, there are five other children, three girls and two boys. Both the boys are normal in all respects, but in the three girls no trace of a knee jerk can be obtained even with reinforcement. There are no other signs of disease except in the fifth child, aged 5, whose feet are in an early condition of pes cavus; in this child the plantar reflex is sometimes of an extensor, and sometimes of a flexor type. She has also a considerable degree of hypermetropia, associated with a divergent strabismus, which is probably due to paresis of the internal recti.

Dr. Head.—*Tremor from a lesion, possibly of the optic thalamus.*

Mary J., aged 21. During the last week of November, 1903, she suffered from momentary giddiness. On December 1 she suddenly became so giddy that she clung to the railings for an hour unable to stand without support; everything seemed double. On reaching home seemed well, but next morning noticed that the right eye was affected, and the right side of the mouth was "drawn." On December 2 she had another attack whilst returning from work. From this time she noticed that the head and right arm showed a tendency to jerk; she spilt her tea, and could not hold her head. A noise like a train whistle was constantly present in her left ear.

Since her admission to hospital in January she has had no further attacks. She has had slight headache only, and has not vomited. She now walks with difficulty, tending to fall to the right. The head is in constant movement, and the movement is mainly rotatory. On grasping the neck it is found that the forcible movements are restitution movements. When she sits in a chair the right leg is occasionally kicked out suddenly; when she stands marked irregular movements of the right arm and leg appear. Closing the eyes makes no difference either to her stability or to the movements. She can now get along without support, but tends to rotate to the right (with the hands of the clock). No paralysis is present in any limb. The knee jerks are brisk, right greater than left. No ankle clonus is present. The plantar reflex gives a flexor response on both sides. Slight weakness of the right external rectus is present; but the noteworthy change in the ocular movements is as follows: When allowed to fix an object the eyes move briskly to the left and to the right, movement to the right being somewhat deficient owing to weakness of the right external rectus. When told to look to the right the eyes either do not move or move irregularly, and she seems to roll them laboriously to the right. Voluntary movement of the face to command is well performed, but when she smiles marked weakness appears over the lower part on the right side. The right half of her tongue moves irregularly when protruded. Movements of the right half of the palate are weaker than the left. Over the whole right half of the face sensation to pain and to the extremes of heat and cold is lost, but cotton wool touch and the appreciation of two compass points are unaffected. Sensation of the remainder of the body is normal. Disc and fundus show no change. She exhibits considerable deafness of the right ear. Taste and smell are unaffected. The sphincters are unaffected.

Dr. G. F. STILL.—*Case of ataxia.*

H. I., aged 15, well till three years ago, when he had a fall from a tree, six weeks later writing became shaky, six months later walking unsteady. He contracted diphtheria and had diphtheritic paralysis subsequent to the onset of his disease.

Present condition.—Intelligent; articulation jerky; irregular movements of the eyes; no nystagmus; paralysis of left side of palate; slight intention tremor of hand; marked incoördination in gait; constant irregular movements of the trunk; knee jerks active, the right is greater than the left; plantar reflex gives extensor response.

Dr. Leonard Williams.

(1) F. C., aged 11, schoolboy. Three years ago his mother first noticed that the patient shook his head and made faces. Recently, twitchings of the upper arms have become pronounced. His grandfather suffered from fits. His father has also been subject to fits, but has apparently been cured by an operation.

The boy is bright and intelligent. He has never had any serious illness, but when younger had rickets and night-terrors.

The jerking movements, which are so fine and so frequently repeated as to give the impression of a tremor, are chiefly confined to the upper arms.

The head is also affected, and, to some extent, the face.

(2) *Generalised tremor associated with tabes.*

J. H., aged 61, printer. Three months ago had an attack of "a peculiar feeling at the heart," which was immediately followed by a "trembling all over," lasting several minutes. The sensation was quite indescribable, but there was no loss of consciousness. The attacks have gradually increased in number, and he now has as many as eight in the day.

He has the Argyll-Robertson pupil; absent knee jerks; Rombergism; impaired sensation to touch and delayed sensation to pain in the feet, with some ataxy. When made to close the eyes tightly he has some difficulty in opening them again. This is especially noticeable on the right ride. (He had ptosis on this side fifteen years ago.)

The tremor of the whole body is quite apparent when he is standing, but not when he is sitting.

Tremors of the hand are well seen when he keeps the forefinger on the nose.

(3) *Tremor of unusual distribution associated with Parkinson's disease.*

J. B., aged 35, a cabinet maker, who has been much in request as an amateur violinist. A year ago he noticed that his left hand became clumsy, and he was obliged to give up the violin. Six months ago he noticed the left leg to drag when he became excited, which at this time he began to do very readily when in company with friends. Shaking on the whole of the left side then showed itself, and is now making its appearance on the right.

He has a pronounced mask and a peculiar reptilian stare, due to the fact that he seldom blinks. He cannot raise his brows, or frown.

There is a fine tremor of the whole head; the eyelids tremble when closed, and the lips when the teeth are shown.

His speech is slow and his voice monotonous, but he has no difficulty in enunciating difficult words.

The tremor in the left hand is very apparent, that in the right less so, but both are unmistakable when he is tested for Rombergism.

The knee jerks are very active; but there is no clonus, and several attempts to elicit any response to plantar stimulation have failed.

Dr. GEORGE JOHNSTON.—*A case of (?) paralysis agitans with intention tremor.*

The patient, a man aged 35, formerly a sign writer, has several symptoms strongly suggestive of paralysis agitans. His general appearance is in favour of that diagnosis. He has the fixity of expression, and the attitude of Parkinson's disease; the arms being somewhat rigidly flexed at the elbows, and the hands held in the interosseous position, with the thumbs opposed to the forefingers. His gait is similar to that of paralysis agitans, and there has been occasionally a tendency to retro-pulsion and latero-pulsion. So long as he is perfectly at rest there is no tremor, or at most a very slight movement of the hands. On any volitional movements of the hands and arms, tremor at once starts, and is more like that of paralysis agitans than that of disseminated sclerosis. There is very slight incoördination in the movements of the arms. This volitional tremor is also present in the lips, tongue and eyelids.

The optic discs are normal. There is no nystagmus. The knee jerks are brisk. There is no ankle clonus, and the plantar reflex is normal.

Dr. E. FARQUHAR BUZZARD.

Boy, aged 10 years, exhibited fine tremor of head, trunk and arms, which was first noticed at 3 years of age and has been constantly present ever since. The patient is not so well developed as other members of the family, is well behaved but sulky, nervous, and backward at school. Presents frequent frown-ing and other facial movements suggestive of some mental defect, but is clean in his habits. Four other healthy children in the family. Full-term child and healthy at birth; walked at 2 years of age. No evidence of other disease of the nervous system.

Dr. WILFRED HARRIS.—*Case of familial tremor.*

A man, aged 73, worked as a compositor until seven years ago. Commenced to show tremor in the arms when aged 40. This very slowly increased in severity, until at the age of 63 the tremor became more general, and rapidly got worse, so that he was unable to feed himself, or to walk more than a few yards. He was treated at a hospital in London, where, according to his statement, the case was diagnosed as paralysis agitans, and was treated with strychnine. Under this treatment he improved considerably, so that at the end of six weeks he was as well as he is now. Since that time he is always easily tired after any exertion, and has to go to bed every afternoon for two hours, and again at eight o'clock at night, or the tremor becomes more severe. With any exertion the tremor of the hands soon becomes excessive, while at rest very little shaking is visible. His writing is shaky, and after writing a few sentences becomes almost illegible. His grip is weak, and he is very short of breath. Reflexes normal. He swings his arms in walking, walks and turns well, and has none of the aspect of paralysis agitans, with a mobile expression. There is no mental deficiency. His brother who is fifteen years younger, has suffered from a similar tremor for the last thirty-three years, and this brother's son has also developed it during the last ten years.

Dr. T. GRAINGER STEWART.

D. B., female, aged 31, married.

Complaint.—Shaking of the arms.

Antecedent history good.

Present illness came on two years ago after a fright. At first the tremor was general, but varied greatly both in severity and in position. During the last year it ceased, but came on again two months ago and was localised about the shoulders.

The tremor is rhythmical in character and consists mainly of a rotatory movement of both humeri. It ceases on voluntary movement and also during sleep. The patient can control the movement for some time.

The examination of the nervous system reveals no signs of organic disease.

At the Clinical Meeting held at 11, Chandos Street, on Thursday, July 14, at 8.30 p.m., the following cases of Muscular Atrophy were shown :—

(1) Muscular atrophy of spinal cord origin.
(2) Peroneal type.
(3) Neuritic type.
(4) Anomalous cases.
(5) Syringomyelia.

(1) Muscular Atrophy of Spinal Cord Origin.

Dr. Henry Head.—*Widespread muscular wasting of both upper extremities with increased knee jerks and flexor toe response.*

Henry N., aged 50, machine printer. For eight months he has noticed wasting of the muscles of the left hand. Three months ago this produced such loss of power that he ceased work; he did not know that his right hand was affected until his attention was called to the wasting at the hospital. At no time has he had any pain.

There is marked wasting of the left upper extremity, and to a less degree of the right. On the left side the supra-spinatus, infra-spinatus, deltoid, and both parts of the pectoralis major are wasted, and there is profound loss of power in the deltoid. Triceps and biceps are wasted, act feebly and show fibrillary contractions. The extensor muscles of the wrist and fingers are much wasted and extension is impossible. There is wasting and loss of power in the muscles of the thumb and the interossei. The extensors of the wrist and fingers, the first dorsal interosseus, do not react to faradism, and react sluggishly to galvanism.

Of the muscles of the right upper extremity the supra- and infra spinatus, pectoralis major, upper part of the deltoid and first dorsal interosseus are wasted, but all these muscles react to faradism.

The legs are unaffected; sensation is perfect, and, except that the tongue shows fibrillary tremors, the cranial nerves are unaffected.

The knee jerks are exaggerated; there is no ankle clonus. The toes go down on both sides and tibial pressure causes a flexor response.

Dr. FERRIER.—*Amyotrophic lateral sclerosis* (*upper arm type*).

J. F., aged 52. Ten years ago both shoulders and arms became rather rapidly weak and began to waste. The weakness continued to progress for about four months. Under treatment the disease became arrested, or at least ceased to progress appreciably till about three years ago, when the arms became considerably weaker, and more wasted. Recently the patient had not been able to walk so well. He had not noticed any change in articulation or difficulty in swallowing.

There is great wasting of several of the muscles round the shoulders, especially of deltoids, supra- and infra- spinati, biceps and triceps. The latissimi dorsi, serrati magni and pectorales are of fair size.

All muscles of the forearms and hands are of fair size and have good power.

Constant fibrillation is present in the wasted musles.

The muscles of the lower extremities are large but not strong in relation to their bulk. *Gait* is natural.

He cannot now whistle as he formerly could, but there are no other cranial nerve symptoms to be connected with this illness.

No sensory changes are present.

The knee jerks are exaggerated, and the plantar responses are of the extensor type.

Till the commencement of this illness he was a painter by trade, but never had wrist-drop or other symptoms of plumbism.

Dr. FERRIER.—*Progressive muscular atrophy* (*? early tabes dorsalis.*)

M. B., aged 46. Slowly progressive wasting and loss of power in hands, arms and shoulders, of two years' duration. Began in right fingers, and five months after onset she noticed some weakness of right arm. Twelve months ago the left hand became similarly affected, and since her last confinement, nine months ago, both arms have wasted very rapidly, and for some months now have been quite useless to her.

She is not aware of any affection of her legs, and has not experienced any difficulty in swallowing or in articulation. There have been no pains or paræsthesiæ.

Her previous health has been good, alcohol can be excluded, but not the possibility of specific infection.

There is extreme wasting of the muscles of both upper extremities and shoulders, including trapezii, supra- and infra- spinati. Rhomboids, levatores anguli scapulæ, and upper fibres of the trapezei remain fairly good. Upper portions of the pectorals wasted more than the lower, latissimi dorsi and serrati magni are of fair size.

There is very little power of voluntary movements at any joint.

No wasting of the lower extremities and the strength of all movements is good, but the muscles are rather toneless. The gait is quite natural.

In addition—the pupils are unequal and scarcely react to light, but they contract on accommodation. The knee jerks are absent, though each extensor quadriceps is strong, and there is a slight degree of hypoalgesia on feet and legs. Romberg's sign, however, is absent, and there is no sphincter trouble.

The electrical irritability of the muscles (both faradic and galvanic) corresponds to the degree of wasting, and no polar change is present.

Plantar stimulation gave flexor responses.

(2) Peroneal Type.

Dr. HENRY HEAD.—*A case of peroneal muscular atrophy.*

George D., aged 30, bag-maker. In 1893, after he had been ill with a bad cold, and lost nearly two stone in weight, he first noticed that the muscles of his legs were wasting. At the same time he lost the grip in both hands, but did not know the muscles were wasted until it was pointed out to him by his doctor. In about two months all wasting ceased, and he went to work as a trunk-maker. A few weeks ago his legs began to waste again, but he thinks his hands are exactly as they have been for the last ten years.

He stands well with slight lordosis, and does not stagger when his eyes are closed. He has difficulty in going up-stairs, and in walking up hill. All the muscles below the knee on both sides are profoundly wasted, but all the movements of the same can be performed, although less strongly than normal. The interosseous spaces are wasted in both hands, especially in the left. Both little fingers tend to be abducted when the hand is at rest.

All the wasted muscles react to faradism, but their irritability to both that and galvanism is diminished, and strong currents must be used to obtain even a small contraction. Sensation is unaffected, the cranial nerves and knee jerks are normal, and the toes go down.

A case of advanced peroneal atrophy.

Joseph A., builder's handy-man. Five years ago he was walking home when his right leg gave way, and he fell. For some little while before this he had noticed "rheumatic pains" down the back of the right leg. Two weeks later his right foot suddenly dropped when he was on a ladder, and has never been right since. Three weeks afterwards his left foot dropped in the same way. A year later he first noticed that his hands were getting thin. Ever since then he has periods in which the muscular atrophy and weakness increase, followed by many months during which he improves somewhat. He thinks that the wasting is always accompanied at first by slight rheumatic pains, and that if these are situated in his legs, it is the legs that waste; recently they have occurred in the shoulders, and the muscles of the shoulder and upper arm are the seat of the wasting at present.

He has always been a temperate man and never suffered from lead-poisoning.

He can only stand with help, but does not fall with his eyes closed. Both feet are dropped and the toes point downward: dorsiflexion is impossible. There is much wasting of the anterior tibial group and of the peronei. The calf muscles are small, but act well. In the thigh the extensor group is weak and wasted. The flexors of the hip act poorly. There is no fibrillary twitching. All the muscles react to faradism except the anterior tibial group on the left side. On the right side faradism causes a flicker only. To galvanism the left anterior tibial group does not react.

In the upper extremities there is marked wasting of all the small muscles of the hand, the extensors of the fingers are weak and the extensor surface of the forearm is wasted. Supinator longus, biceps, triceps, deltoid are flabby, diminished in size and act feebly. Supra- and infra- spinatus are wasted.

All the muscles of both hands and forearms react to faradism. In the left arm deltoid, biceps and triceps do not react; in the right arm the triceps does not react, and the biceps and deltoid react with a slow contraction to a strong current.

Sensation, reflexes and cranial nerves are normal.

(3) Neuritic Type.

Dr. HENRY HEAD.—*Muscular wasting from brachial neuritis.*

Walter D., aged 47, engineer. December 27, 1903, he first noticed tingling in the right arm. This gradually increased until, by the end of January, it had become acute, especially at night. About the beginning of March, 1904, he noticed wasting of the muscles of the right hand.

All the interossei and the muscles of the little finger of the right hand are much wasted; the inner thenar group are also wasted. The right arm and fore-arm are smaller than the left, but there is no wasting of any isolated group of muscles. All the muscles react normally to faradism and to galvanism.

He complains of pain down the ulnar side of the arm and fore-arm, and over this area there is increased susceptibility to prick, to heat, and to cold; light touch is unaffected.

The reflexes are unchanged and there are no further abnormal signs in the nervous system.

Dr. HENRY HEAD.—*A case of atrophy of the lower portion of the trapezius.*

Alfred E., aged 49, a gardener. About December, 1903, his wife noticed that the bone in his back projected. For six months before this he had noticed "rheumatic" pains in the right shoulder. Since the beginning of 1903 these pains have ceased and the condition of the shoulder has slowly improved. He is sober and has never had gout. He shows paralysis and wasting of the lower portion of the right trapezius. The senatus acts well. The wasted muscle reacts to faradism and does not show a slow reaction to galvanism. There are no other abnormal signs in the nervous system. His urine is normal.

(4) Anomalous Cases.

Dr. ORMEROD.—*? Early syringomyelia or neuritis.*

F. H., aged 14. About two months ago the child began to complain of pins and needles and numbness in the right hand, and soon showed evidence of weakness of the right hand and fingers. Since then this weakness has been slowly progressive. About a month later the left hand and arm became affected in the same way, but the weakness has not reached the degree present in the right.

There has been no noticeable weakness of the lower extremities or affection of the cranial nerves.

Status presens.—Some general wasting of the muscles of the hands, greater on the right than on the left, and of the fore-arms, especially along their ulnar borders.

On the right the movements of the fingers and wrist are practically lost, and are very weak and limited on the left. Flexion of each elbow is fair, though neither supinator longus contracts, but extension is relatively more feeble.

The movements of the shoulders are good, and the lower extremities are unaffected.

No fibrillation has been observed in the wasted muscles, and there is no tenderness on pressure.

There has been complete loss of pain sensation in the right fingers and the ulnar border of the hand, and considerable diminution of thermal sensation to the middle of each fore-arm.

There is no history of pain, and the paræsthesiæ have disappeared.

The knee jerks are brisk, and the plantar reflexes of the flexor type.

There has been very slight improvement since she came under observation.

Dr. James Taylor.

A. W., aged 25, came on account of weakness in right hand and arm. This has become much worse lately, although the patient says she believes it has been present since she was 4 years of age.

There is weakness of the small muscles of the hand, with marked cyanosis of the whole hand. There is nystagmus, slight lateral curvature and extensor response from each sole.

The case is presented as probably one of syringomyelia, but remarkable on account of the complete absence of sensory impairment to any form of stimulus.

(5) Syringomyelia.

Dr. Farquhar Buzzard.

A woman, age 59. Previous health good. Two children born dead, five others alive and healthy.

Four years ago, after a strain, noticed left hand gradually becoming weak and wasted. About the same time drooping of the left eyelid was noticed.

Two or three years has suffered pain in arms of a gnawing character.

One year has had at times a tight feeling round waist.

Present condition.—Atrophic palsy of left hand. Left pupil and palpebral fissure smaller than right. Left pupil does not dilate well to shade. A zone of dissociated anæsthesia around trunk, extending from just above umbilicus to groin. Some thermanæsthesia on the arms. Abdominal reflexes absent. Legs strong, and present no spasticity or atrophy. Slight lateral curvature of spine.

Dr. WILFRID HARRIS.

A man, aged 20, has suffered for the last eighteen months from constant numbness and tingling in the right arm and leg, while for the last five months he has noticed frequent attacks of giddiness, and headache on the right side of the forehead. The giddiness is generally worse in bed, but during the day he often tends to fall towards the right side. No vomiting or convulsion. Right eye often gets bloodshot. Has never burnt or cut himself without feeling the ordinary amount of pain. No history of syphilis.

Present state.—There is some weakness of the right grip, with partial right hemianæsthesia to all forms of sensation, especially to pain and heat, on the right side of the head and face, arm and shoulder, as far down as the nipple, and commencing again at the groin, including the whole right lower limb, but excluding the inside of the cheek, tongue, gums and palate, and the trunk below the nipple. During the last month, under treatment by sinusoidal current, the area of loss of sensation on the right lower limb has disappeared. On the left side there is an area of analgesia with partial loss of pain and temperature sense above the second rib, corresponding to the fourth cervical area, though the analgesia is less deep than that on the right side.

For the last three months he has been hoarse, and there is now complete abductor paralysis of the right vocal cord, and of the right half of the soft palate, with marked anæsthesia of the right tonsil and right side of the pharynx. There is coarse lateral nystagmus, most marked to the right side, but the cranial nerves are otherwise normal. There is complete atrophy of the middle and lower trapezius and of the rhomboids on both sides, with atrophy of the left levator anguli scapulæ, and less complete of the left upper trapezius, and of the left erector spinæ. Both sterno-mastoids normal. The knee jerks are equal and brisk, the left triceps jerk is greater than the right, and there is a bilateral extensor plantar reflex.

There is a well-marked mid-dorsal scolio-kyphosis, with the convexity to the right, and the left scapula hangs much lower than the right.

The case is of interest on account of the recent onset of vertigo, falling to the right, nystagmus and headache, with slight right-sided weakness, being suggestive of a cerebellar lesion, such as tumour, on the right side. The palsy of the right vocal cord and soft palate, however, were more suggestive of a nuclear lesion, and was the first clue to syringomyelia, while on stripping the patient, the irregular distribution of the right hemianæsthesia, the muscular atrophy, and the area of dissociated sensation of the left fourth cervical area confirmed the latter diagnosis.

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Original Articles and Clinical Cases.

ON THE CELLS OF THE SPINAL GANGLIA AND ON THE RELATIONSHIP OF THEIR HISTOLOGICAL STRUCTURE TO THE AXONAL DISTRIBUTION.

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INTRODUCTION.

THE importance of the spinal ganglia in physiology and in pathology has long made the structure of these bodies an object of research for neurologists. The striking and familiar features are the marked variation in size of the component cells, and the very different appearances these cells show when stained in an appropriate manner. Attempts have been made to correlate these differences with the fact that the cells are the nutritional centres for nerve fibres distributed to very different tissues of the body. Our communication is a further endeavour in this direction.

Since the discovery of Ranvier that the cells of the ganglia gave origin to an axon dividing immediately into a peripheral and central process, some new facts of importance have been ascertained.

Firstly.—The elaborate studies with the Nissl method made by many observers, which have culminated in the admirable work of Lugaro (1) have shown that a classification of the cells may be made on account of the different appearances they present when stained by this method, and that the types thus recognised may be found in all animals.

Secondly.—Numerical observations, chiefly from the Chicago laboratory of Professor Donaldson, have brought out interesting and suggesting facts, notably the excess in number of cells over fibres in the posterior roots.

Thirdly.—It has been shown that besides the usual cell with the T-shaped axon of Ranvier there are in the ganglia other kinds of cells. Dogiel (2) [1] from preparations made by Ehrlich's methylene blue method, has described multipolar cells. He states that they are scanty in number, and that their processes may or may not have a myelin sheath. He was unable to follow their processes beyond the ganglion. The existence of these cells has been confirmed by several observers. Dogiel has also described cells whose axones are short and divide very often, ending in a pericapsular and pericellular basket situated around the ordinary type of cells. They therefore resemble the Golgi cell of type II. met with n other parts of the nervous system, and may be looked upon as relay cells. They belong to the smaller class of cells and are not numerous.

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[1] In Bayliss' Paper (*loc. cit.*) there is a diagram which shows plainly the types of cells described by Dogiel.

The ganglion is therefore a more complicated structure than was formerly thought; it is a nucleus of termination as well as of distribution. In this paper we give the result of observations chiefly based on a more detailed method of using the "axonal reaction" than has previously been attempted, and endeavour to show that they suggest an answer to some of the problems indicated by the foregoing sketch.

METHODS USED.

Our experimental lesions have all been made on cats, and no results accepted unless healing without visible reaction occurred. In each case the animal was allowed to live fourteen days, except in the last experiment.

The method of intra-vitam fixation was used in all cases. The vascular system was first thoroughly washed out with normal saline, and afterwards Mann's fixing fluid of corrosive sublimate, picric acid and formalin allowed to run through the vessels. The fluids were used at a temperature of 38° C. After complete saturation of the tissues with this fluid, the ganglion was removed as speedily as possible and placed in the same fluid for an hour. We found the best results were obtained by a rapid method of embedding, the ganglion being passed through the several dehydrating and clearing agents at a temperature of about 38° C., and remaining in each of these and the final paraffin bath for from 10 to 15 minutes. In this way we obtained many preparations in which the fixation seemed to be perfect. Of dyes, we found toluidin blue used alone to be the best, and, if neutral balsam is used, the stain is permanent for at least many months.

As a rule a counter-stain with erythrosin was not used, since this dye, unless very faint, prevents the finest granules staining with the blue, a fact alluded to by Lugaro and by Orr and Rows (3).

The position of the ganglion examined was ascertained after counting the ribs, which in all instances numbered thirteen.

Serial sections of $\frac{1}{150}$ mm. thickness were cut usually through the whole extent of the ganglion, and consecutive series of ten examined in order that the whole of any one cell might if necessary be observed.

With regard to the chromatolysis produced by the various lesions, we are satisfied that in the normal animal there are no cells which cannot after careful examination be distinguished from cells in a state of chromatolysis. If a cell is cut through the axon hillock it can readily be distinguished by the normal characteristics of the remaining Nissl granules. Other deceptive appearances are cleared up by adopting the rule never to consider cells in which the nucleolus is not clearly visible. It is only amongst the small dark cells that reasonable doubt arises, but after careful staining by toluidin alone, and attention to the characteristic features described by Lugaro, especially the condition of the nucleus and its surrounding stained protoplasm, we are convinced that the margin of error is small.

Certain control experiments were made when necessary, and will be mentioned later.[1] The measurements of the sizes of cells and nerve fibres mentioned in the following pages were obtained as follows:—A Leitz drawing ocular was used, and having obtained by means of the usual micrometers the exact magnification produced by a given lens, with the draw tube of the microscope at 160 mm., the size of the image thrown on to a drawing paper was accurately measured by means of a steel rule graduated to $\frac{1}{10}$th of a millimetre.

By dividing this number by the ascertained magnification the size of the cell was obtained.

This method enabled us to readily measure a large number of cells, and is strictly accurate. The measurements given all relate to cells in which the nucleolus was clearly visible, and included the whole protoplasm as far as the innermost part of the capsule.

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[1] At the Oxford Meeting of the British Medical Association, we had the pleasure of demonstrating our specimens to Dr. Orr, whose studies on the ganglion cells are so well known, and he kindly allows us to say that he agrees with the statement that in the normal animal it is very exceptional to meet with cells which can be confused with a true chromatolytic change, and also that in his opinion the appearances shown in our specimens could only have been the result of an experimental lesion.

THE SIZES OF THE CELLS IN GANGLIA OF DIFFERENT REGIONS.

For this purpose we selected the second cervical, the eleventh dorsal, and the seventh lumbar ganglia, and 1,000 cells from each ganglion were measured in the manner described.

The second cervical receives fibres from the skin and small muscles, the eleventh dorsal in addition receives afferent visceral fibres, whilst the seventh lumbar has no visceral connections, but receives the sensory fibres from the large hamstring muscles.

The results obtained may be best represented graphically (fig. 1). The diameters of the cells were measured to the nearest even μ, and the curves given were obtained in the usual way from the actual measurements, which are not represented in the diagram. The divergence of the observed from the calculated numbers was not great, and left no doubt as to the interpretation.

In general terms the curves from the cervical and dorsal ganglia show great similarity in the proportion of cells of different sizes, which is strikingly confirmed by the facts that the greatest number in each case measure about 40 μ and that this figure also represents the mean diameter (actual figure = 41 μ). The curve of the lumbar ganglion is widely different, and is a double one, the second part representing added cells of larger size than exist in either the cervical or dorsal regions. The mean diameter of all the cells in this case being brought up to 55 μ. These added cells, as will be seen from the experimental results mentioned later, represent the large clear type, and are the cells in connection with the sensory organs, the muscle spindles of the large hamstring muscles.

They thus represent the added elements of the first sensory neuron consequent on the development of the limbs.

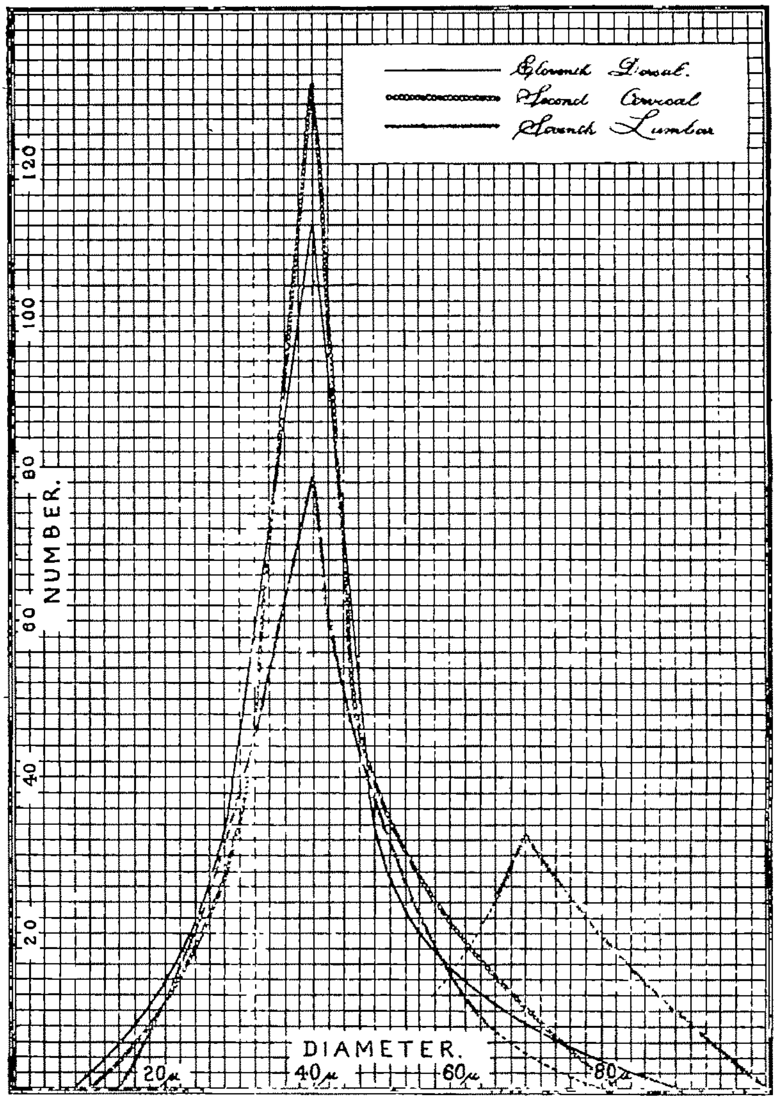

FIG. 1.

Curves showing the sizes of 1,000 cells in each instance from the second cervical, eleventh dorsal, and seventh lumbar ganglia. The similarity of the numbers of cells of different sizes in the second cervical and eleventh dorsal is shown. In the seventh lumbar the facts brought out are, (1) the smaller number of cells of medium diameter, (2) the added cells of large diameter (consequent on the connection of this region with large muscles of a limb).

The Types of Cells in the Ganglia, and a Review of Some of Lugaro's Conclusions.

It has been mentioned that Dogiel and others have described multipolar cells and cells of the Golgi type II. as present in the spinal ganglia. The Nissl method, whilst incapable of demonstrating those cells, has formed the basis of other methods of classification. The more important papers are those of Lenhossék (4), in 1895-96; Held (5); Marinesco (6), 1897; R. A. Flemming (7), 1897; Cox (8); van Gehuchten and Nelis (9), 1896. In addition to these papers Lugaro has published a long series of researches dating from 1896 to the present time, and some of his conclusions will be shortly noticed. The classification established by Lugaro's observations in animals has been extended to man by Orr and Rows (10), who also in their paper give a review of the work of previous observers.

Lugaro's observations were made on several kinds of animals, the rabbit, dog, cat and guinea-pig. In all these he recognised the same five types of cells.

(1) Large clear cells with granuliform chromophile elements scattered almost equally throughout the protoplasm, with a slight increase towards the cell periphery. The nucleus is large and clear, and there is a definite perinuclear and peripheral clear zone.

(2) Large and medium sized cells, with very fine chromophile elements, which are larger, however, at the periphery of the cell. Other characteristics as in type I.

(3) Small dark cells, with very fine chromophile elements, larger around the nucleus with which they are in contact. The fundamental substance and nucleus are diffusely coloured.

(4) Small and medium sized clear cells, with chromophile elements somewhat large but few in number. The nucleus is clear and separated from the chromophile substance by an achromatic zone.

(5) Large clear cells, with elongated chromophile elements lying concentrically around the nucleus in parallel

planes. The nucleus is always excentric, whereas in the other types it is almost uniformly central.

Orr and Rows (*loc. cit.*), John Turner (10) and ourselves, working after the publication of the major part of Lugaro's work, confirm in the main this classification, and hence the important position is reached that the same types of cells may be recognised in different mammalia.

Without entering into minute histological details, it is necessary that we should make a few observations regarding this classification, which we think may be simplified. Lugaro's types I. and II. are recognised by all, but we consider the sub-division an unnecessary refinement not always obvious. We find the size of the cells with this structure varies, within wide limits, from 35 μ to 100 μ. The peripheral clear zone has been recognised since E. Müller's paper (11). It is not a distinct membrane, but is merely cell protoplasm, from which chromophile granules are absent. In perfectly fixed specimens it is not readily visible, but becomes obvious when there is any cell retraction. Lenhossék even expressed the opinion that it might be an artefact. Increase, however, in this zone is not necessarily due to imperfect fixation, for we can confirm R. A. Flemming's observation that after section of the axon the zone is relatively increased. Cassirer (12) also notes this. The perinuclear clear zone exists in many of the larger cells, but not in all. Lenhossék says it is seen mostly in the largest cells. We subsequently allude to these types as the *Clear Cells*. Lugaro's type III. may be called *Obscure Cells*; they have been described and figured in all the papers we have consulted from Walther Flemming (13) onward. They are the most numerous cells in the ganglia and vary greatly in size, from 10 μ to 56 μ in our analyses, and hence include some of the smallest of the cells. Lugaro's type IV. we propose to call *Coarsely Granular Cells*; they are readily recognised under the low-power of the microscope, and are very scantily represented, they have a fairly uniform size between 35 μ and 50 μ. Lugaro's type V. has been called by that writer the Vorticose Cell. It is stated by Lugaro and Orr and Rows to be very rare. We have been unable to clearly recognise it, and hence must disregard it in a scheme of classification, a position also adopted by Turner. Lastly, amongst the smallest cells, in addition to the obscure cells, we find some in which the network is less dense, consisting of either fine or scanty largish granules. The fundamental protoplasm is not diffusely coloured, and the nucleus is always proportionately very large and usually central. These cells never exceed 25 μ in diameter, and a capsule cannot always be seen. We allude to them as the *Smallest Clear Cells.* (Fig. 2.)

Classified in this way our analyses give the following percentages :—

	2nd Cervical.	11th Dorsal.	7th Lumbar.
Clear cells	25·5 ...	21·9 ...	35·5
Obscure cells	68·1 ...	72·6 ...	56·8
Coarsely granular cells ...	4·3 ...	2·5 ...	3·7
Smallest clear cells... ...	1·9 ...	2·6 ...	4·2

Lugaro in his extensive research has shown that in the different types of cells the alteration following an axonal lesion and the subsequent fate of the cell vary, both in the rate and kind of change. Hence a cell may be recognised as belonging to a given type throughout the series of changes constituting reaction and repair. He claims that the different morphological types of cells in the sensory ganglia may be considered as specifically distinct, both from an anatomical and physiological standpoint.

THE GANGLION CELLS OF THE HUMAN FŒTUS.

Dr. A. S. Griffith and one of us have examined the spinal ganglia in the human subject at the third, fourth, sixth and eighth month of intra-uterine life. At full term the several types of cells present in the adult could be recognised, but at an earlier age this differentiation was absent, and the cells resembled considerably the smallest clear cells described above. It was found that the chromophile elements first appeared at the periphery of the cell. At the fourth month the cells were found to be of about the same size, and often showed doubl[illegible]li[illegible]ey were commonly grouped

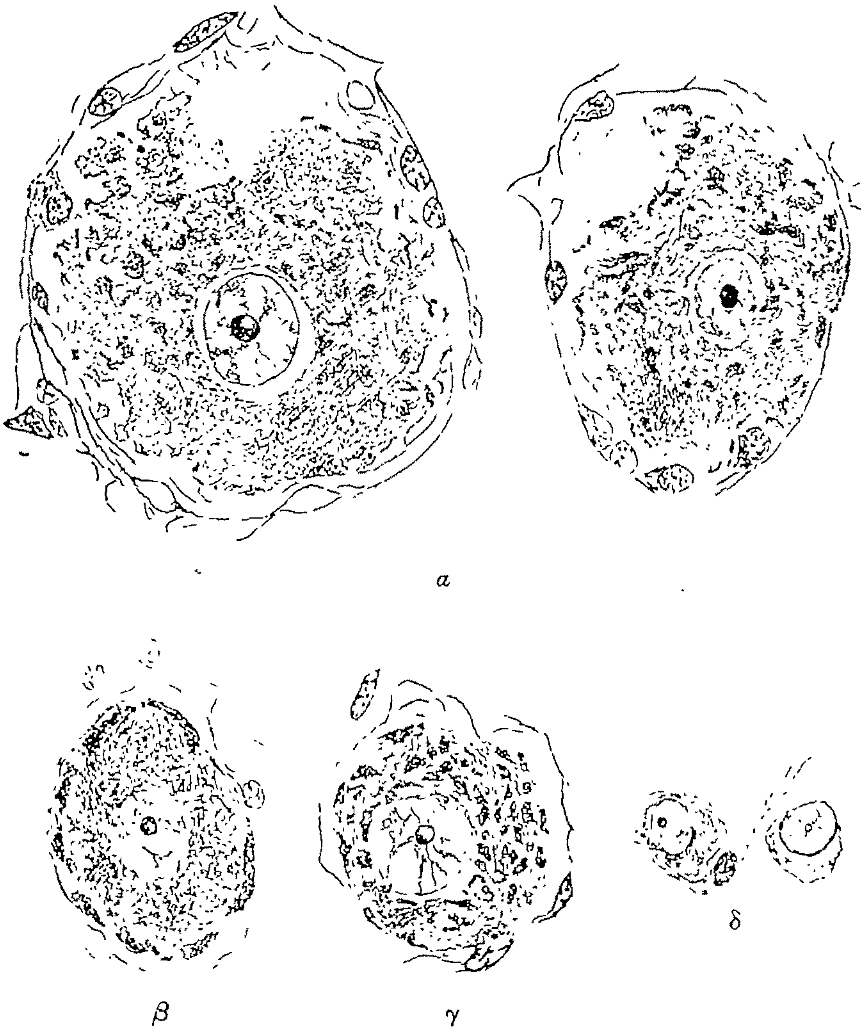

Fig. 2

Normal cells drawn from the seventh lumbar ganglion. Leitz $\frac{1}{12}$. Drawing ocular.

(α) Clear cells (86 μ and 70·3 μ).
(β) Obscure cell (53 μ).
(γ) Coarsely granular (43·8 μ).
(δ) Smallest cells, obscur[illegible]d cl[illegible]7·2 μ and 15·8 μ).

PLATE I.

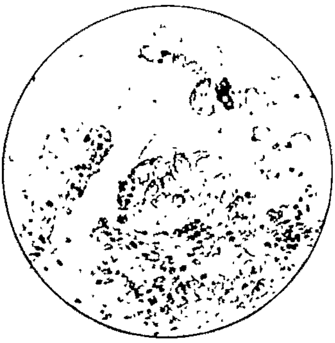

FIG. 1.

Micro-photograph of a section of a lumbar ganglion of the human fœtus at the third month. Zeiss $\frac{1}{8}$. Showing the embryonic type of cells in a colony.

in colonies of four or six. The colony, and not the individual cells, was surrounded by a capsule. At the sixth to the seventh month the commencement of the formation of types was first clearly seen. (Plate I.)

Observations similar to this have been made by Van Biervlet (14) in man, and by Dall'Isola (15) and Olmer (16) in animals.

Lugaro states that he has observed these embryonic types as constituting the normal character of the cells in the frog and the tortoise.

The type of reaction is, as shown by our figures, one of central chromatolysis, and hence there is justification for the statement originally made by Van Biervlet that the morphological characters of cells in the reactive phase are met with in cells undergoing development, and are also seen in lower forms of philogenetic development.

An interesting pathological observation by Sibelius (17) indicates that in congenital syphilis the cells of the full term fœtus also show a retardation of development as evidenced by the presence of double nucleoli, the formation of colonies, and the absence of a capsule around the individual cell.

THE CELLS WHICH GIVE ORIGIN TO AFFERENT FIBRES IN THE WHITE RAMI.

It is known from physiological experiment that every white ramus contains afferent fibres. This can also be readily demonstrated by division of the spinal roots between the ganglion and cord, and subsequent examination of the ramus. If sufficient time be allowed—forty-two days or more—all the fibres derived from the spinal cord degenerate, those remaining intact must have their cells of origin in the spinal ganglia. The white ramus is then stained in 1 per cent. osmic acid, and may be examined either after teasing, or it may be embedded and cut in transverse section.

In this way Sherrington (18) determined the number of afferent fibres in the tenth thoracic white ramus of a small cat to be 150, the diameters varying from 12 μ to 1·5 μ.

In a personal communication Dr. Anderson informs us that the twelfth thoracic ramus in a cat was found to contain 374 afferent fibres, of which forty-two were large (8 to 11 μ), seventy-five medium (6 to 8 μ), and 157 small (4 to 6 μ).

Langley and Anderson (19) found 1,100 afferent fibres out of a total of 3,500 fibres in the pelvic splanchnic or nervus erigens. Similarly, after the division of the anterior and posterior roots of the first and second thoracic nerves in a cat, we allowed forty-eight days for any fibres having their cells of origin in the spinal cord to undergo complete degeneration. The white rami were then dissected out, stained in osmic acid, and cut at various levels in transverse section

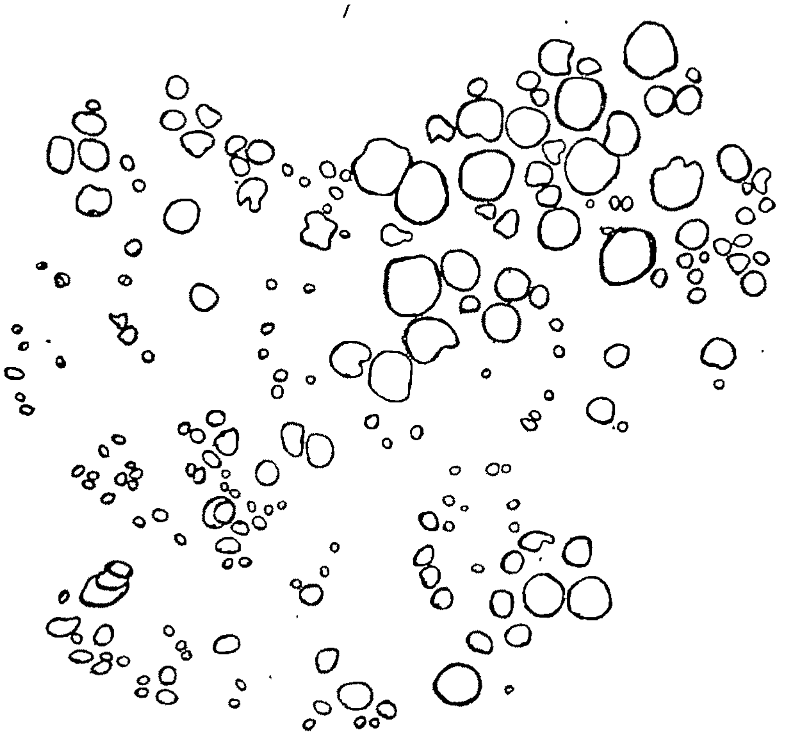

FIG. 3.

Afferent fibres of the second dorsal white ramus, which remain undegenerated 48 days after division of the anterior and posterior roots. Leitz $\frac{1}{12}$. Drawing ocular.

(fig. 3). There were found in the first ramus 136 sound fibres—

5	with	a diameter	under	2 μ
53	,,	,,	between	2 to 4 μ
9	,,	,,	,,	4 to 6 μ
27	,,	,,	,,	6 to 8 μ
34	,,	,,	,,	8 to 12 μ
8	,,	,,	,,	12 to 15 μ

In the second ramus there were found 192 sound fibres—

5	with	a diameter	under	2 μ
90	,,	,,	between	2 to 4 μ
38	,,	,,	,,	4 to 6 μ
32	,,	,,	,,	6 to 8 μ
23	,,	,,	,,	8 to 12 μ
4	,,	,,	,,	12 to 14 μ

Thus in each case more than half the fibres are over 4 μ, which is about the maximum size to which efferent sympathetic fibres attain. With regard to the few fibres which were over 12 μ, some of these were distorted owing to the contraction of the remaining tissue of the nerve, and, as the longest diameter was measured, their real size was probably under that given. The fibres also in some cases were cut rather obliquely. This point should be mentioned as fibres over 12 μ have not previously been found in the sympathetic system.

A few additional remarks may conveniently be made here. Gaskell (20) describes and figures in the dog a communicating branch between the first and second dorsal nerves, which runs along with and in the same connective tissue sheath as the second white ramus; we did not find this condition in the cat.

Edgeworth (21) has by dissection shown that large fibres (7·2 to 9 μ) are present in all the white rami of the dog, but the number of such fibres would appear from the figures given above to be considerably more numerous than he estimated; thus he only found ten large fibres in the first dorsal ramus and fourteen in the second.

We have next to enquire as to the variety of cells in the spinal ganglia which give origin to afferent fibres in the white rami.

We are again indebted to Dr. Anderson for the description of an operation which exposes the stellate ganglion of the thoracic chain between the heads of the first and second ribs. The operation is almost bloodless, and, beyond a little separation of the muscles attached to the scapula, involves only the teasing away of the first intercostal muscles. The ganglion and the first and second dorsal rami running to it can be seen on the pleura, and the desired lesion easily made (fig. 4). The experiments made consisted in one case of section of the second dorsal ramus, in another of a median division of the stellate ganglion, and in three of its total removal. Before describing the results of these observations, it should be mentioned that a series of control experiments was made, in which the stellate ganglion was exposed but not touched, and the condition of the first, second and third spinal ganglia examined.

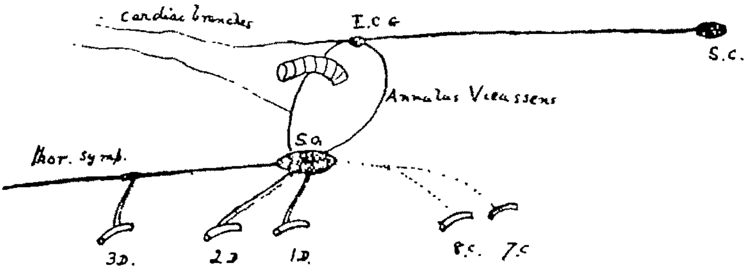

FIG. 4.

Diagram of the connections of the stellate ganglion. The three first dorsal nerves are represented with the white (continuous line) and gray (dotted line) rami, and the two lower cervical nerves with gray rami only. The other connections are indicated in the figure.

Briefly the results were as follows :—

Controls 1 *and* 2.

Stellate ganglion on the left side exposed but not touched. First, second and third dorsal spinal ganglia on both sides examined.

In the first experiment the three ganglia of the right side did not show any cells other than normal. On the left side the first and third ganglia also appeared normal. In the second ganglion a series of twelve sections containing about 1,200 cells and taken at different regions of the ganglion, showed nine cells of the small dark type, in which there was eccentricity of the nucleus, with some scantiness of the central granules; in two of these cells there was an accumulation of Nissl granules around one surface of the nucleus, a feature which Lugaro has alluded to as being characteristic of chromatolysis. We also found one cell of a larger type which we were in doubt about. In the second control experiment we did not find even this small number of cells whose condition was doubtful.

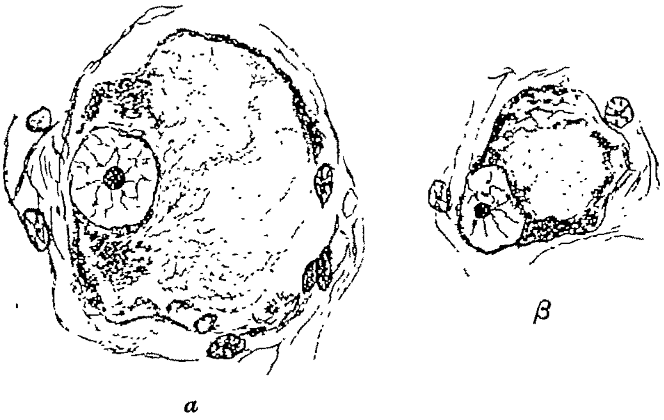

FIG. 5.

Altered cells in the second dorsal spinal ganglion after section of the corresponding rami communicantes. Leitz $\frac{1}{12}$. Drawing ocular.
(*a*) Clear cell (62·4 μ).
(β) Obscure cell (39·8 μ).

Control 3.

The second dorsal ramus cut, first and third spinal ganglia examined. Our note reads: "Sections remarkably well stained, all details shown. No cells can be seen which are at all comparable with those showing chromatolysis in the second spinal ganglion.

Since in the experiments following the nerve lesions, we adopted a strict criterion of chromatolysis and rejected any examples which appeared doubtful, we do not think our findings can be attributed to any other result than the nerve lesion.

Experiment 1.

Second dorsal ramus of the left side cut; condition of cells in the corresponding spinal ganglion studied.

The whole of the ganglion was cut into sections, and many of these examined.

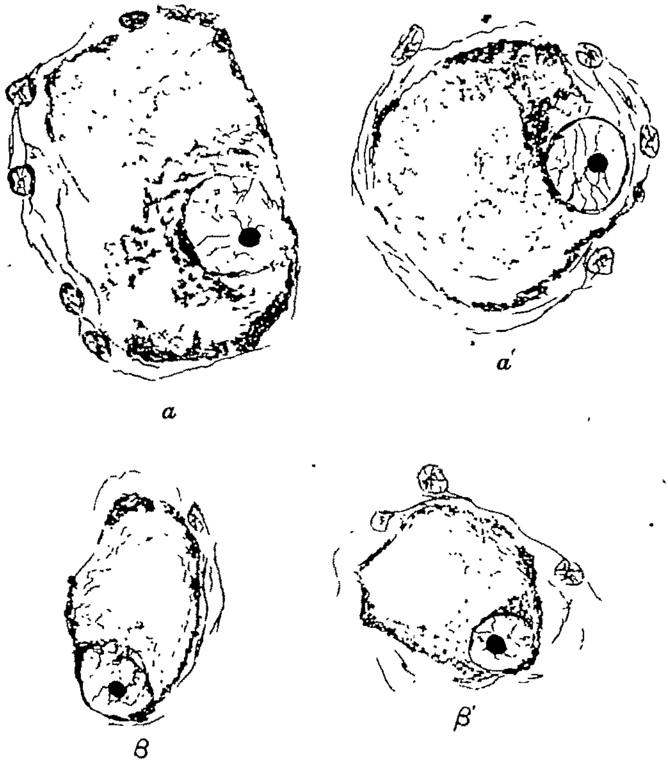

FIG. 6.

Cells showing chromatolysis in the third dorsal spinal ganglion, after lesion of the stellate ganglion. Leitz $\frac{1}{12}$. Drawing ocular.
(α) Clear cell (65 μ).
(α′) Clear cell (68 μ).
(β) Obscure cell (43 8 μ).
(β′) Obscure cell (37 μ).

A number of cells, being a very small percentage of the total number forming the ganglion, were found to show complete chromatolysis (fig. 5).

Of these thirty-five were measured.

6 had a diameter between 50 and 55 μ
10 ,, ,, ,, 40 ,, 50 μ
19 ,, ,, ,, 30 ,, 40 μ

Experiment 2.

Central piece of stellate ganglion removed on the left side. The first to seventh dorsal ganglia were examined. Evidence of chromatolysis was found in each ganglion, as far as and including the fifth dorsal (fig. 6).

The protocols give the following figures (a section contained 70 to 100 cells).

First dorsal.—Thirty sections examined. Twenty examples of chromatolysis found; of these fourteen were small cells, six rather larger.

Second dorsal.—Twenty-five sections examined. Twenty-three examples of chromatolysis found, sixteen small cells, six rather larger, and one larger again.

Third dorsal. —Forty-five sections examined. Forty-one examples of chromatolysis found; twenty-seven small cells, twelve rather larger and two larger again.

Fourth dorsal.—Forty sections examined. Seven examples of chromatolysis; four small cells, three larger.

Fifth dorsal.—Forty-two sections examined. Three examples of chromatolysis found; two small cells, one larger.

Sixth and seventh dorsal.—These ganglia did not contain any cells showing chromatolysis.

Measurements were made of forty cells taken from different ganglia.

7 had a diameter between 50 and 55 μ
14 ,, ,, ,, 40 ,, 50 μ
19 ,, ,, ,, 30 ,, 40 μ

Experiments 3, 4 *and* 5.

The stellate ganglion of the left side was removed after careful section of all its branches. The first to seventh dorsal ganglia were examined.

It is unnecessary to give the protocols of these experiments, which yielded results exactly comparable to those just cited.

Remarks.

The Wallerian degeneration has furnished unequivocal evidence that the viscera are furnished with afferent fibres, having their cells of origin in the spinal ganglia,[1] and we have shown that the Nissl "axonal reaction" is capable of indicating these cells. Both methods show that the number of afferent fibres is small, and that the proportion of cells in the spinal ganglia thus connected with the sympathetic system is very small, perhaps barely reaching at most 2 per cent. It is not difficult to say that in our experiments nearly all the cells belong to the smaller obscure cells. This is shown by the comparison of their size and also because they can easily be recognised to undergo the features of chromatolysis, which are characteristic for that variety of cell. They do not, however, belong to the smallest type of cell, since none of those measured by us were less than 30 μ. It can also be stated that they show no features by which they can be distinguished from other small, dark cells, whose axones have a different distribution. Cells quite similar have been known to occur in all the spinal ganglia, and we shall later show that they undergo the same kind of chromatolysis after section of a cutaneous nerve. Some doubt remains as to the type to which the largest cells, those of 50 μ, belong. The smaller members of the "clear type" approach in size some of the obscure cells, and Lugaro has shown that in the condition of chromatolysis they may greatly resemble each other. Unaltered cells of the coarsely granular type could be seen in all our specimens. This cell is also present in regions of the cord which do not give origin to sympathetic fibres. It has been suspected by other observers that the small, obscure cells give origin to visceral fibres. Thus Head and Campbell (22) point out that in herpes the ganglia most affected are those in connection with white rami, and mention that such ganglia contain a larger proportion of small cells. The number of afferent visceral fibres being, however, so small, this reason cannot be an adequate one, and, further, the small, dark cells are present in all ganglia. To demonstrate the relationship direct experiment is necessary. We shall show that the large, clear cells are largely those which give origin to fibres distributed to the sensory structures of muscles, and it appears that the largest of the myelinated fibres in the sympathetic system arise from similar cells. In this connection there is the important observation of Anderson (23), who has shown that large, myelinated, splanchnic fibres develop their myelin simultaneously with large, somatic fibres, the one terminating in Pacinian bodies of the viscera and larynx, the other in Pacinian bodies, muscle spindles, and end organs connected with deep somatic structures. Similarly, the finer medullated fibres which terminate in the skin, and which are connected with pressure and painful sensations especially, are myelinated contemporaneously with the fine set of medullated, splanchnic, afferent fibres which, he argues, supply the epithelial structures of the viscera, and which would endow them with common sensibility. Our observations show also that both sets arise from a cell of similar histological structure.

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[1] This view is accepted by British physiologists. Conclusive evidence is furnished by the fact that "after intraspinal section of a nerve just peripherally of the posterior root ganglion, either all, or all but a few, of the medullated fibres in the white rami degenerate, and that after section of the sympathetic or of the splanchnics, or of the inferior splanchnics, no degenerated fibres are present in the white rami." (Langley, "The Autonomic Nervous System," Brain, 1903, page 22.) It seems desirable to state this fact in view of the results obtained by Onuf and Jos. Collins (*Arch. of Neurology and Psycho-pathology*, vol. iii., 1900). In an account of their work read before the American Neurological Association, May 27, 1898, and given in the *Journ. of Nervous and Mental Diseases*, vol. 1898, page 661, those authors conclude that "most of the afferent (sensory) fibres of the sympathetic nerves do not originate from the cells of the spinal ganglia, as Köllicker claims; on the contrary, they must have, in accordance with Dogiel's view, their cell of origin within the ganglia or plexuses of the sympathetic system." In this abstract no details are given of the methods used, and we have been unable to obtain the more extensive account.

Little is known of the distribution of the afferent fibres in the upper dorsal white rami. This could be ascertained by removal of the spinal ganglia, and subsequently tracing the course of the degenerated medullated fibres. We have not yet attempted this. It is known, however, that none pass into the cervical sympathetic, and clinical evidence, especially adduced by Head (24), points to their distribution to the heart and lungs. The augmentor cardiac fibres pass out by the second to the fifth anterior roots, and Head considers the sensory part of the arc is represented in the same segments, or is of wider distribution. He says that the pain attributable to pressure in the left ventricle finds itself mainly distributed to the cutaneous areas supplied by the second and fifth dorsal nerves. Though, owing to the limitation of the method of axonal reaction, too much importance cannot be attached to the fact, it is interesting to note that the fifth dorsal spinal ganglia was the lowest in which we succeeded in finding evidence of changes after lesion of the stellate thoracic ganglion. This experiment indicates that afferent fibres arise as low as the fifth dorsal, run up the thoracic chain, and pass through the stellate ganglion and out by the annulus of Vieussens. That the annulus of Vieussens contains afferent fibres has been shown by physiological experiment by Langley (25), and Edgeworth (26) traced large fibres in it, passing with the cardiac branches of the inferior cervical ganglion. The afferent nerve to the aorta appears to be the depressor, as shown recently by Köster (27), and by Tschermak (28). Since the splanchnics receive afferent fibres as high as the fourth dorsal and perhaps higher, it would appear that the same spinal ganglion may supply both thoracic and abdominal viscera, a fact of interest in connection with the overlapping of the "sensory areas" of referred pain in diseases of the stomach and lungs (Head).

THE CELLS OF ORIGIN OF THE AFFERENT FIBRES TO MUSCLES.

Sherrington (29) first showed that in a muscular nerve trunk from one third to one half of the myelinated fibres are from cells of the spinal ganglion, and that their diameters varied from 2 μ to 20 μ, or that some of these fibres are a hundred times larger than others. He asks, "Does a difference at all corresponding with this exist between the sizes of the cells in the spinal ganglion?" He finds that the diameters of the cells vary from 100 μ to 20 μ, or taking the cells as of spheroidal figure, some have a volume 125 times that of others. The assumption is that large fibres arise from large cells.

We have studied the changes which take place in the spinal ganglia after section of a purely muscular nerve, viz., the hamstring nerve.

Sherrington states that the nerve in the cat contains 4,512 fibres, of which 1,456 are afferent. In three instances we exposed the nerve at the top of the thigh, and a small piece was removed. The sixth, seventh, and eighth post thoracic spinal ganglia were removed on the fourteenth day, and subsequently examined. Obvious chromatolysis of some cells was found in each case (fig. 7).

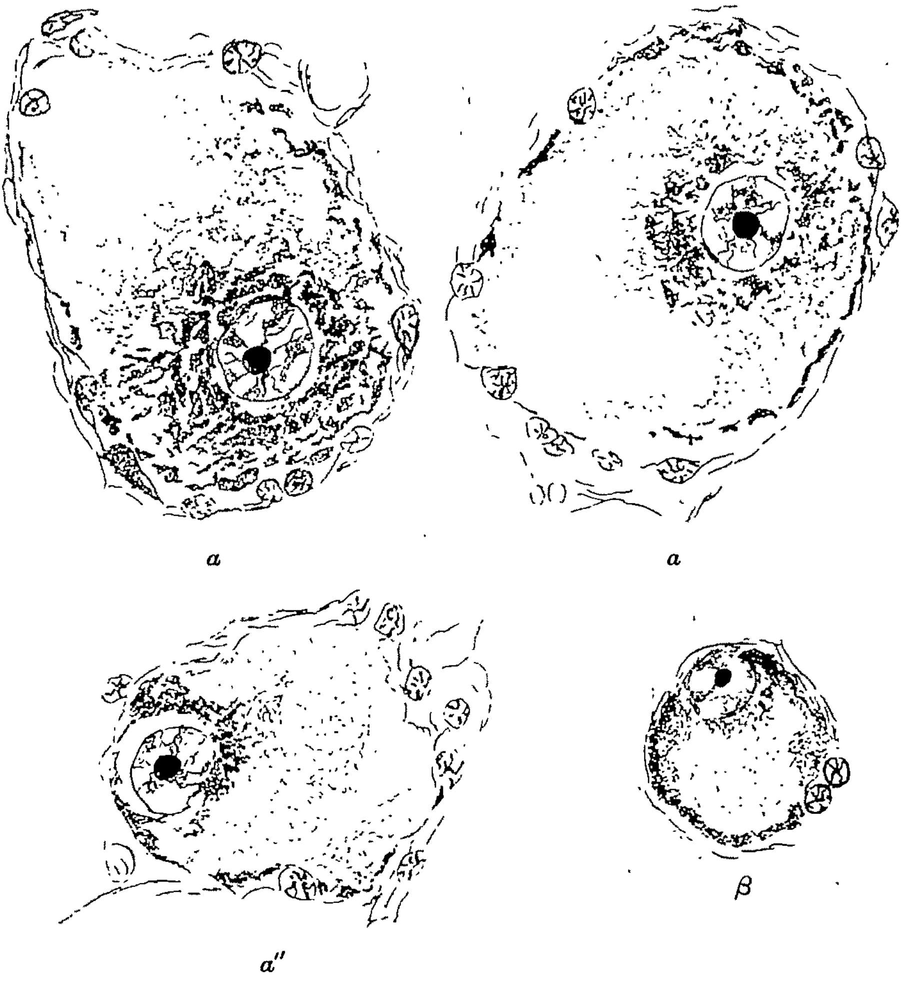

FIG. 7.

Altered cells from the seventh lumbar spinal ganglion after section of the "hamstring" nerve. Leitz $\frac{1}{12}$. Drawing ocular.
(α, α', α'') Clear cells (90·3 μ, 79·7 μ, 57·7 μ).
(β) Obscure cell (36·2 μ).

Of fifty cells accurately measured, the smallest was 32 μ in diameter, the largest 93·5 μ.

11 had a diameter between 30 and 40 μ
17 ,, ,, ,, 40 ,, 60 μ
14 ,, ,, ,, 60 ,, 80 μ
8 ,, ,, over 80 μ

Another series gave—

65 cells with a diameter over 60 μ
43 ,, ,, ,, between 50 and 60 μ
52 ,, ,, ,, ,, 30 ,, 50 μ

The largest cells therefore are those in which the change is most seen, a fact to be considered in conjunction with Sherrington's statement that of the afferent muscle fibres the majority belong to the class whose diameter is 12 μ or more.

The figure shows that the large clear cells are most readily affected. All the largest and most of the medium sized cells showing chromatolysis belong to this type. The obscure cell is the other type showing change.

Many examples of the coarsely granular cell were noticed intact, and the smallest cells of the ganglion, viz., those up to about 25 μ, did not appear changed. From Sherrington's observations we know that only fibres of 7 μ or more are distributed to the muscle spindles, and hence the cell of origin of these is the large clear cell, the remaining cells accounting for afferent fibres to muscle structures having some other kind of end organ.

CELLS OF ORIGIN OF AFFERENT FIBRES DISTRIBUTED TO THE SKIN.

The long saphenous nerve was cut in the upper third of the thigh, and the fifth and sixth lumbar ganglia examined. In an examination of eighteen sections taken from the sixth lumbar ganglion, eighty-five altered cells were found, of which fifty-four belonged to the obscure type, thirty-one to the clear type (fig. 8).

Of forty cells accurately measured—

8	had a diameter between	30	and	40 μ
19	,, ,, ,,	40	,,	50 μ
5	,, ,, ,,	50	,,	60 μ
5	,, ,, ,,	60	,,	70 μ
3	,, ,, ,,	70	,,	75 μ

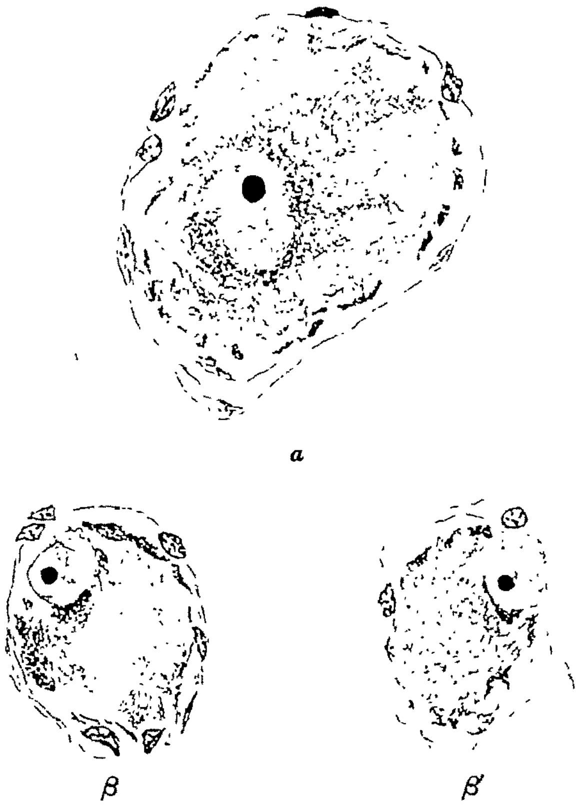

FIG. 8.

Altered cells in the sixth lumbar spinal ganglion after section of the internal saphenous nerve in the middle of the thigh. Leitz $\frac{1}{12}$. Drawing ocular.

(α) Clear cell (69 μ).
(β, β′) Obscure cell (48·8 μ and 41·1 μ).

Sections of the nerve stained with osmic acid showed that the diameters of the fibres varied from 1·95 μ to 15 μ.

Here as in the preceding experiment no cell with a diameter of under 28 μ was found altered, and the coarsely granular cell also did not seem to have been affected. The characteristic large size of the chromophile elements could not be recognised in any of the altered cells, and on the contrary many intact forms of this type were seen.

It thus appears that cells of the obscure type and those under 53 μ in diameter gave origin to the majority of the fibres, the clear cells accounting for the remaining fibres of larger size. The results are in general similar to those obtained in experiments on the visceral fibres, with the difference that a rather greater portion of fibres take origin from the clear cells.

ON THE SMALLEST CELLS IN THE GANGLION, AND ON THE COARSELY GRANULAR CELLS.

The Smallest Cells.

In all the preceding experiments we have been unable to find evidence of chromatolysis in cells of less than 26 μ in diameter.

These very small cells belong either to the obscure or the clear types.

What is the cause of this failure to react?

It may be suggested that since in the preceding experiments the nerves have been cut at some distance from the ganglia, the small cells do not react because they are connected with axones which leave the nerve trunk very soon. The nerves of recurrent sensibility have a short course beyond the posterior ganglion, and would escape section in the lesions made by us.

Also it must be remembered that the axonal reaction is probably more intense the nearer the lesion is made to the cells of origin.

We have therefore observed the result of a lesion immediately distal to the ganglion.

This can be done by exposing the cauda equina, and we have cut the ninth post-thoracic nerve 2 to 3 mm. beyond the ganglion.

After nine days we find evidence of alteration in the great majority of cells. The only cells remaining intact were the smallest cells of under 26 μ. It is unlikely that in this lesion even the nerve fibres of recurrent sensibility would have escaped section.

Lugaro has performed a similar experiment, viz., division of a nerve in the cauda equina, and whilst he does not state the results of the experiment with great precision, implies that practically all the cells show change, and hence he considers that the method of axonal reaction does not support the view that there exist cells in the sensory ganglia whose axones ramify in the ganglion itself, that is, independently of the peripheral nerve.

As thus stated we cannot fully accept this conclusion. Is a small cell peculiarly resistant to damage to its axon? On the contrary, Lugaro's experience and our own is that it very readily undergoes chromatolytic change. Does the very small cell represent the multipolar or "relay" cell described by Dogiel and others? This supposition we have also to reject, chiefly on account of its small size. Dogiel's measurement gave a much greater size. We suggest the explanation is to be found in the observations of Donaldson (30) and his colleagues, to which we rather fully refer.

Hatai (31) has shown that in the white rat the number of cells in a ganglion remains constant at various periods of age from birth to adult life. Averaged from different ages, he gives the following figures as the number of cells in the ganglia of three regions: Sixth cervical, 11,140; second lumbar, 8,867; fourth thoracic, 7,306. Whilst, however, the total number of cells remains constant, both he and Donaldson agree that the relative number of large and small cells varies. With the increase in age the small cells diminish, the large ones increase. This fact they have observed in all regions. In other words, the small cells grow to become large ones. The small cells, therefore, are considered to be in an immature condition and to have no axones, and, though they may later acquire them, many remain permanently immature. Hence a distinction is made between cells in "reserve" and cells in "commission."

Two sets of facts are brought forward to support this contention. First they find that in the newly-born rat there are in the dorsal roots immature fibres, distinguishable by the feeble staining of their myelin, and that as the animal becomes mature both the total number of root fibres increase and the proportion of immature to completely-formed fibres diminishes. Thus in the newly-born rat in the sixth cervical root they find 1,998 fibres, of which 48 per cent. are immature, whilst in the adult animal they find 4,227 fibres, of which only 1·2 per cent. are immature. Secondly, the figures given above of the number of cells in various ganglia show that there is a great excess of cells over fibres. Such excess of cells over root fibres has been previously observed by Hodge, Bühler, Gaule and Lewin, and their results are quoted by Hatai.

Our observations have all been made as the result of a peripheral lesion. Hence, assuming these figures to be correct, and the most careful methods were adopted to ensure accuracy, we have to ask if the same excess of cells exists with reference to peripheral fibres. In other words, what is the proportion of peripheral fibres to root fibres? Dale (32) in the adult cat finds they are practically equal. Holl, Freund, and Stienan also only find a slight peripheral excess. The results of Hardesty (33) indicate that the equality does not exist in all kinds of animals, as in the frog (rana virescens) he found a marked excess of peripheral over central fibres.

The result, however, of these observations of Hardesty, Donaldson, and Hatai, leads to the conclusion that the number of cells in any ganglion is in excess of the peripheral fibres, and that therefore one assumption is that all the cells of the ganglion do not give off peripheral axones.

We consider that these findings explain why the smallest cells do not give the axonal reaction. They are immature nerve cells, and have at least no fully-developed axon. We find their number to be in the second cervical, 7·6 per cent.; in the eleventh dorsal, 13·5 per cent.; and in the seventh lumbar, 13·5 per cent.

The Coarsely Granular Cell.

As stated in the preceding section, the members of the

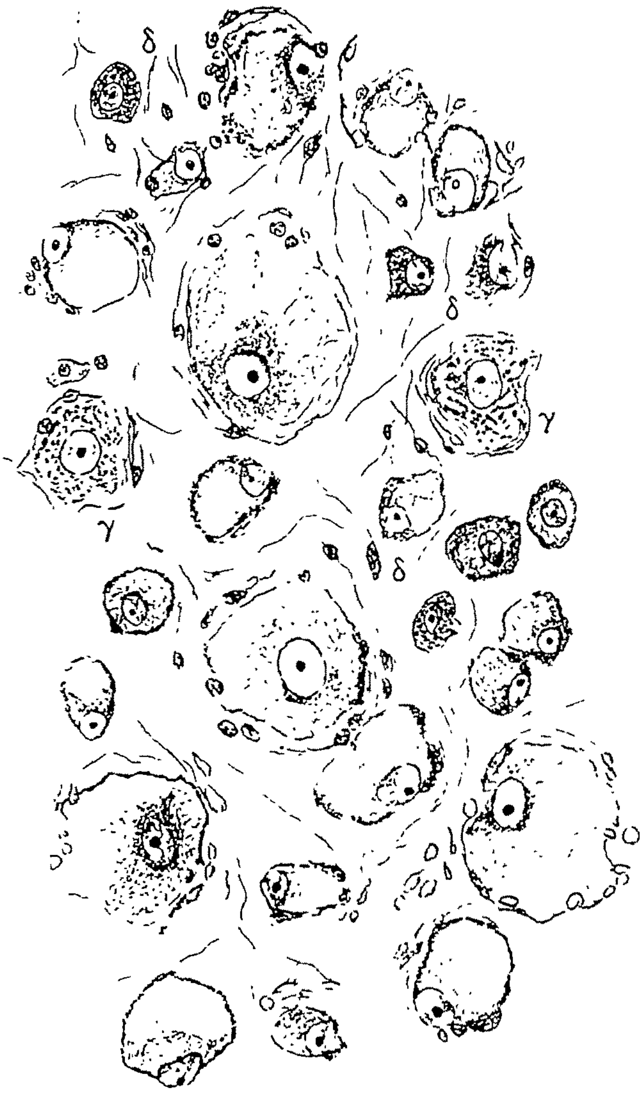

FIG. 9.

Showing cells in the first dorsal spinal ganglion after section of nerve immediately distal to the origin of the rami communicantes. All the cells drawn exhibit chromatolysis except γ coarsely granular cells and δ the smallest cells.

coarsely granular type were found intact in all experiments, except the one in which the lesion was made in the cauda equina. In this case they shared the change common to all other types except the smallest cells of the ganglion.

How resistant they are to a lesion of the peripheral nerve trunk is shown by an inspection of fig. 9, which is drawn from the first dorsal ganglion, and shows the changes following a lesion immediately distal to the origin of the sympathetic ramus. In this case all fibres peripheral to the ganglion had been cut, except the white ramus and the primary dorsal division. The resulting chromatolysis was very extensive, but intact coarsely granular cells were found as well as the smallest cells.

We have no further evidence to advance as to the significance of this cell. It is a very well-defined type, having in a great majority of instances a diameter of between 40 and 50 μ; its chromophile granules are nearly as large and discrete as those of anterior cornual cells of the spinal cord.

The negative evidence obtained points to it being not the cell of origin of fibres distributed to the skin or viscera or muscle structures.

Its great resistance to change may, perhaps, be due to its axon not extending beyond the ganglion, and hence the cell would belong to the class described by Dogiel, *i.e.*, relay cells.

It may also be mentioned that Bayliss (34) has shown that there are vaso-dilator fibres in the posterior roots, that these fibres have their cell of origin in the spinal ganglia, and that the cells belong to the ordinary class having the $\perp$ axon.

SUMMARY OF CONCLUSIONS.

(1) The size of the cells in the ganglia is a function of the diameter of the nerve fibre, and the difference in appearance of the chromophile elements has a dynamical significance. These elements represent material which is used up by the essential trophoplasm, which presides over the nutrition of the nerve fibre.

(2) At the region of the limb plexuses, a number of large

cells appear in the ganglia, which are the cells of origin of the fibres to sensory muscle structures.

(3) Lugaro's classification is confirmed in the main.

The cells met with are best described as (*a*) clear cells; (*β*) obscure cells; (*γ*) coarsely granular cells; (*δ*) smallest clear cells.

(4) The large clear cell and the obscure cell give origin to the fibres distributed to the skin, viscera and muscles. Of these the largest cells are exclusively connected with fibres from the muscle spindles. Large somatic and large splanchnic fibres have origin from cells of the same type, the large clear cell; small somatic and small splanchnic fibres also from cells of the same type, the obscure cell.

(5) Afferent fibres to the thoracic viscera arise from the first five dorsal ganglia. The number of such fibres in one instance was found to be 136 in the first dorsal, 192 in the second white ramus.

(6) The smallest cells in the ganglion, under 26 μ in diameter, whether belonging to the obscure or clear type, are immature cells, and have no fully-developed axon.

(7) The coarsely granular cell is highly resistant to the axonal reaction, and may be a "relay" cell, but sufficient evidence is not forthcoming as to its significance.

(8) The cells in the early embryo have the appearance of the smallest type described in the text. In the newly-born human subject the types present in the adult can be recognised. The morphological characteristics of the cells of early embryonic life are also met with in the reactive phase.

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ON CERTAIN TREMORS IN ORGANIC CEREBRAL LESIONS.

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THERE is perhaps no symptom in neurology whose anatomical basis or the physiology of whose production is less understood than those involuntary movements which, though varying in their character and accompanying conditions, are collectively known as tremors. Their occasional association with various gross organic lesions of the central nervous system has been recognised and described for years, but, on the one hand, indefinite and incomplete clinical characterisation, and on the other unsatisfactory and often defective description of the localisation and nature of the pathological lesion, has made a study of the cases unproductive of tangible results.

Owing to the loose way in which the term tremor has been employed in the literature of the subject, it seems advisable to offer at once such a conventional definition as will serve as a basis for the more detailed description of its several characteristics. I would suggest that the term tremor be used to denote a clinical phenomenon consisting in the involuntary oscillation of any part of the body around any plane, such oscillations being either regular or irregular in rate and in amplitude, and due to the alternate action of groups of muscles and their antagonists. If such a definition be accepted it becomes obligatory in describing the symptom to note the regularity or irregularity of the range and of the rate of its component oscillations. Here, too, emphasis must be laid on the importance of detailing exactly what movements are comprised in a particular tremor. A tremor may be simple or compound; in the former case only one group of muscles and its antagonists being involved, in the latter the resulting movement being the effect of the action of several groups and their antagonists. For instance, flexion and extension of the fingers may coincide with pronation and supination of the forearm. In such cases the study of the condition evidently resolves itself into the study of the individual simple movements.

The graphic representation of the oscillations as first employed by Gowers, or of the muscular contractions by some form of ergograph, is advisable, generally almost necessary, for a complete study of all clinical tremors, and direct tracing of the movements is when possible preferable to tambour records as being more accurate. This method has been employed in the examinations of all the cases here recorded as well as in that of a number of other cases which have been carefully studied for the purpose of contrast and comparison. When the tremor is compound it is necessary in taking a tracing to mechanically check all but the one movement which is being studied, or to adopt some device to exclude the complications introduced by other movements.

In addition to the detailed description and graphic record of the movements of which the tremor is composed, it is important to note the influence of volitional movement, of attempted voluntary inhibition, of sleep, and of psychical rest and activity.

The cases recorded in the paper presented clinical symptoms of considerable similarity, and the evidence thus afforded suggested a common site for their pathological lesions, a suggestion which was verified in the two cases examined *post mortem*. The aim of the paper is to give a concise clinical description of the cases, to ascertain from direct and indirect evidence the nature and position of the lesions, to attempt to exactly define the essential factor in the lesion, to discuss the possible pathogenesis of the tremor, and finally, to compare and, contrast the cases with identical or similar ones collected from the literature.

I must first express my indebtedness to Dr. Buzzard, Dr. Hughlings Jackson, Dr. Ferrier, Dr. Ormerod, Dr. James Taylor, and Dr. Risien Russell, for permission to use the cases I have observed under their care in the National

Hospital, and to Dr. Farquhar Buzzard for having kindly supplied me with the description of the lesions which he, as pathologist, found in the cases which were examined *post mortem.*

Case 1.—R. E., the first case observed, was a healthy-looking woman of 59 years, with an excellent family history, and no history of previous illness, who came under the care of Dr. Ormerod in July, 1903. Four months previously she suddenly and without loss of consciousness became unable to walk and found her left arm useless. She was taken to an infirmary, where she slowly regained the power of walking, but has never since been able to get about as she formerly could. She also complained of the left arm having remained "useless," though she was able to move it about quite well, that it became quite unsteady when she attempted to do anything with it, and that when even at rest it frequently shook. She had not been able to read since the onset of this condition, and has had diplopia. There was no headache, vomiting, or other symptom in the history of her illness which could raise the suspicion of cerebral tumour.

On examination her visceral organs were found normal, but her pulse tension was unnaturally high, though no other evidence of renal disease could be detected. There was no psychic impairment, but she was abnormally emotional, and frequently burst into tears or laughter without adequate cause. Her vision was fair and the visual fields were uncontracted, but she was unable to read owing to paralysis of accommodation. There was also complete paralysis of the upward movement of both eyes, and the downward movements were defective, but the range of lateral excursion and convergence remained good. There was also marked bilateral ptosis, with compensatory wrinkling of the forehead in the attempt to look up. Both pupils were inactive to light stimulation, and contracted very little and sluggishly on convergence. Since then there has been slight return of the upward movement of the left eye, but the palsy of the levators of the right remains complete. The range of downward movement is much increased; at present it is almost normal. Her face was stiff, fixed and expressionless, and there was little change of expression as she conversed, but the voluntary movements were all perfect, and the associated movements good. There was, however, very little movement as she attempted to smile, and those which occurred when she laughed or wept were slow and stiff. The speech, too, was monotonous and expressionless. The mask-like facial expression and the monotonous and toneless articulation gave to the case, as many who saw her remarked, the appearance of paralysis agitans.

When she first came under observation there was definite paresis of the left limbs, with some rigidity, brisk deep reflexes, and an extensor plantar response on the same side. The right limbs were then normal in every respect, and of good power. The left hemiparesis soon disappeared, and the plantar extensor response was replaced by one of the normal flexor type, but the slight degree of rigidity has remained unaltered.

In walking both limbs and trunk were held stiff, without any swing or play of movement; she shuffled along with short steps in an uncertain manner, and turned *en bloc*, so that her gait, though neither reeling nor ataxic, appeared affected out of all proportion to the slight impairment of power of the lower extremities. The left hand was generally held in the interosseal position, the fingers extended at the distal and partially flexed at the metacarpo-phalangeal joints, with the thumb opposed to the index finger. There was no other peculiarity in the position of the limb. The whole arm was slightly rigid; there was almost uniform and constant resistance to passive movement through its whole range. The voluntary power of the various movements was, as compared to the corresponding of the right limb, only little feebler.

Except when the limb was at perfect rest and so supported that each segment bore its own weight, there was constantly slow clonic tremor of one or other part of it, but more marked at the distal than at the proximal joints. Frequently the adducted fingers were flexed and extended at their basal joints, the index finger rubbing against the opposed thumb, which was in synchronous tremor. Often it was flexion or extension of the hand at the wrist, or flexion and extension, but more frequently pronation and supination, at the elbow. Movements were less frequently visible at the shoulder.

Similar tremor was occasionally to be observed in the left lower extremity, and, as in the arm, more frequently at the distal than at the proximal joints. It most often took the form of extension and flexion of the foot.

The character of the tremor was constant, no matter in which portion of either limb it was observed. It was remarkably regular in rate, and varied very little in amplitude, so that the movement of the limb, as long as only one group of muscles was involved, was essentially regular and rhythmical, but as more than one group was frequently at one time in action, the resultant movement was often a compound tremor. It was slow in rate, from 2½ to 3 oscillations per second, and of considerable amplitude. (Tracing I.)

It always ceased during sleep and when the limb was allowed to lie at rest, so supported that each portion bore its own weight. If any part of the limb was allowed to hang passively (as the hand when the limb was held up by the forearm), the tremor immediately began at the most proximal joint of the unsupported portion. It was increased by any excitement or agitation on the part of the patient, and by movement of the opposite limb. The movements could only be inhibited for a very short time. Voluntary movements of the limb were complicated by wide irregularities of the intention tremor type, but there was no other affection of co-ordination. There was no alteration of any form of sensation. The patient's condition still, now fifteen months after the onset of the illness, remains practically unaltered.

Diagnosis.—The existence of such definite ocular palsies indicates disease in the mid-brain, and the sharp differentiation of the movements affected points to direct involvement of the oculo-motor nuclei, the lesion extending more to the right than to the left of the middle line. The sudden onset of the condition indicates a lesion of vascular origin.

Case 2.—A. R., a woman aged 37 years, came under the care of Dr. Ormerod in November, 1903, complaining of uselessness of the right limbs, affection of gait and double vision. Her family history and previous health were good. Four months previously she had fallen down a flight of stairs, bruising and cutting the left side of her head over her ear. She lay unconscious for two hours, and had to keep her bed for a few days. Then she noticed diplopia on looking below the horizontal, the images being one above the other. Since then, too, her right arm has been more or less useless; "she didn't seem to have the power to do anything with it;" she has not, for instance, been able to use it in dressing her children, though there was no loss or limitation of any movement. Three weeks after the accident the right upper extremity began to shake, and all its movements became very irregular. The tremulousness of this limb has gradually increased till the present.

It was a month after the accident that her walking first was affected, and since then it has rather rapidly become worse. She also complains of the right leg shaking. There have not been any aphasic symptoms associated with this right-sided affection. She spontaneously remarks that she has been very low-spirited, and has frequently wept without cause during the illness.

The patient is a dull, stupid woman, poorly educated but extremely emotional, frequently weeping without adequate or any cause, and in the same way occasionally bursting into uncontrolled laughter. Her visceral organs were normal. She occasionally suffers with headache, but there was no optic neuritis or history of vomiting.

Her distant vision was normal, but there was paralysis of accommodation of the left eye, so that she could not read with it as she formerly could. On looking up, the left eye evidently did not move so well as the right, and there was diplopia, due to weakness of both elevators. The downward movement of the left eye was also affected, and the patient had diplopia on looking below the horizontal plane.

The lateral movements were unimpared, there was no ptosis and the pupils, which were equal, reacted well to light and on accommodation. There was no appreciable difference between the two sides of the face when at rest or in voluntary movement, but in emotional expression the right side moved distinctly less than the left. Both sides of the face were, however, rather fixed and expressionless as she conversed. The musculature of the limbs was fairly well developed for a woman of her size, and the left limbs seemed normal in all respects. On the other hand there was slight rigidity and relative weakness of the right limbs, but no limitation of the range of movement. There was also a marked slowness in starting every voluntary movement, and in exerting the full possible force, which was then not well maintained.

Both right limbs were affected by tremor when they did not lie at perfect rest with their muscles completely relaxed, but it also came on when each portion of the limb was not so supported as to bear its own weight. Thus, it was rarely to be observed as the limbs lay in bed and the patient was quiet and unexcited. It might occur at any joint, but in both arm and leg was more frequent at the distal. The most constant movement was flexion and extension of the hand or of the fingers at the metacarpo-phalangeal joints, but there was also often adduction and abduction of the fingers, and occasionally the thumb might be moved against and simultaneously with the forefinger. In the leg flexion and extension of the ankle was most frequently observed, also similar movement of the toes, but various movements at the more proximal joints were often present.

The tremor was generally compound, that is, it was rarely limited to one group of muscles and their antagonists, so that the limb was as a rule simultaneously moved at two or more joints.

It never persisted for long in any group of muscles. The clonic contractions of each group of muscles involved were essentially regular in rate but relatively slow, from 3 to 3½ per second, and the range of the movements was more or less regular but considerable, *i.e.*, the tremor was slow and coarse. It ceased everywhere during sleep and was increased in amplitude but not in rate by excitement or agitation and by forcible movement of the fellow limb. The patient was unable to check it voluntarily except by allowing the limb to fall relaxed in perfect rest. There was marked intention-tremor of both right limbs, *i.e.*, abrupt deviations from the direct line of movement increasing towards the completion of the voluntary act.

When admitted to hospital she was unable to walk alone, though the strength of each lower limb was good enough to permit it, but she was soon able to get about the ward without assistance. Then she walked with short shuffling steps and body bent forward and held remarkably stiff. Though there was practically no tendency to fall, she was very reluctant to walk without support or assistance, being apparently afraid to trust her weight on her right foot, though she could to order stand on it alone for a considerable time. She did not reel or stagger or drag either foot. There were no definite sensory changes. The deep reflexes were exaggerated in the right side and extension of the great toe resulted from stimulation of the right sole.

Diagnosis.—In this case the hemiplegia alternans superior, right hemiparesis with affection of part of the left oculo-motor nerve, indicated a lesion in the left side of the mid-brain, which, from the mode of onset and course, was probably of vascular origin.

Case 3.—R.·T., a strong and robust-looking man of 21 years of age, was admitted under the care of Dr. Buzzard, in February, 1904.

His health had been excellent until eight weeks previously, since when he had suffered with intense occipital headache and vomiting, and had staggered like a drunken man, always stumbling and falling to the left. Some time later his left arm became unsteady when he tried to use it, and his sight began to deteriorate. Diplopia also developed, and he was noticed to have a squint, "the left eye turned in." He also complained of attacks of giddiness for some weeks, in which he felt inclined to fall to the left, and had the sensation of translation of objects in front of him from left to right (clockwise).

He was found on admission to be in a very weak, almost collapsed, state, and was very drowsy and inattentive, and consequently difficult to examine.

His hearing was not affected, though he complained of tinnitus in his left ear; his vision was very much deteriorated, and he had extremely intense optic neuritis evidently of considerable duration. There was complete paralysis of the left external rectus, and considerable weakness of the right. The inward and downward movements of the eye-balls were unaffected, but the upward movements of each eye were considerably limited. Coarse nystagmus resulted on lateral movements, slower and coarser in the attempt to look to the left than to the right. There was slight paresis of the left side of the face, but no interference with the functions of the other cranial nerves.

The left limbs were definitely weaker than the right, and were, in addition, wildly ataxic in voluntary movements. He was unable to walk alone, with support reeled and staggered about. All deep reflexes were absent, and plantar stimulation gave flexor responses. Sensation was not affected in any part of the body.

From these symptoms a tumour of the cerebellum, chiefly in the left lateral lobe, was diagnosed, and Sir Victor Horsley, who was called in, decided to make an attempt to remove it two days after the patient's admission to hospital. There was found to be enormous increase of the intracranial pressure, but no tumour could be found in exploring the cerebellum. As, however, the pressure suddenly fell and the brain, which was exposed, collapsed and some fluid escaped on cutting into the left lateral lobe of the cerebellum, it seemed probable that a cyst had been ruptured. The patient's condition improved very much after the operation, and most of the neuropathic signs began to disappear.

Three weeks later, however, he again became drowsy and weaker, and a new symptom slowly developed. From this time till his death, about three weeks later, there was almost constantly tremor in all four limbs. It only ceased during deep sleep, or when the muscles were quite relaxed and the limbs so supported that each segment bore its own weight. The tremor commenced at once when any of the muscles were put in tension or brought into action, as when part of either limb was allowed to hang over the edge of his bed, or when it was held up in the observer's hand. It was easily made out that the series of oscillations were due to alternate contraction of the one group of muscles and its antagonists, *i.e.*, that the movements comprised a true tremor. If the limb were left in a suitable position it seemed as if the tremor would persist indefinitely. It was forcible and not easily checked, and showed very little tendency to overflow into other groups of muscles when passively stopped in one set. As two or more movements might simultaneously occur, the resultant movement of the limb was often compound. The upper extremities were more affected than the lower, and the left limbs very much more than the right. In the upper limbs the movements most frequently observed were flexion and extension or pronation and supination of the elbows, flexion and extension of the wrists, and flexion and extension or abduction and adduction of the fingers, which generally remained extended at interphalangeal joints. In the lower extremities flexion and extension of the ankles and similar movements of the toes were the most frequently present, but there was also often similar oscillations of the thigh round the hip joint and of the leg at the knees.

The tremor was in every part regular in rate, about five to six oscillations per second, and the rate did not seem to vary, no matter what part of any limb was affected. It may be described as coarse, *i.e.*, of large amplitude, and the range of oscillation often changed in a more or less rhythmical manner, *i.e.*, the range of successive oscillations would slowly increase to a certain point and then slowly decrease (Tracing II.). Consequently the tremor must be described as irregular in range. He was unable to voluntarily inhibit the tremor, and its amplitude was increased by any excitement or agitation, or by forcible movement of the opposite limbs. The finer voluntary movements, too, were complicated by ataxia and irregularity of the intention-tremor type, so that he scarcely found it possible to bring his hand to his face.

With the increase of the drowsiness and general feebleness all his limbs became weaker, the left more so than the right, and at the same time rigid or rather stiff, so that they offered considerable resistance to passive movement. Some general loss of sensation on both sides of the body was also observed, and later Babinski's sign was obtained on plantar stimulation.

Some difficulty in swallowing developed towards the end, his articulation became almost unintelligible, and he slowly lost all power of movement of the eye balls. As the general weakness progressed the tremor became more feeble, yet it persisted in some degree till his death.

Diagnosis.—When the patient first came under observation there was every justification for the diagnosis of a left-sided cerebellar tumour, but the later bilateral spastic symptoms and involvement of the oculo-motor nerves pointed to extension of the growth to the mid-brain.

At the autopsy a large tumour was found lying in the dorsal half of the mid-brain which extended into the thalamus, destroying the dorsal and medial nuclei of each side in their caudal halves more extensively on the left than on the right side. There it extended a short distance into the hippocampal gyrus, but on neither side reached farther forwards than the middle commissure.

Posteriorly its end lay in and almost filled up the upper half of the fourth ventricle, and extended into and involved the left lateral lobe of the cerebellum and the neighbouring portion of the middle lobe.

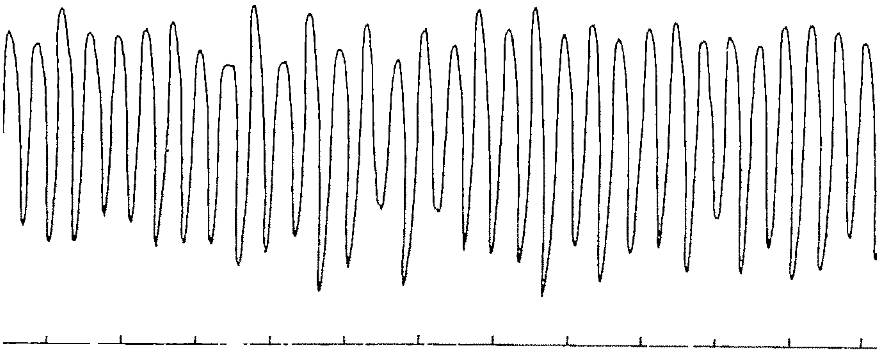
The tumour was well limited and did not infiltrate the structures neighbouring those it destroyed. It did not reach nearer than 1 cm. to the pyramidal tracts in any part of their course.

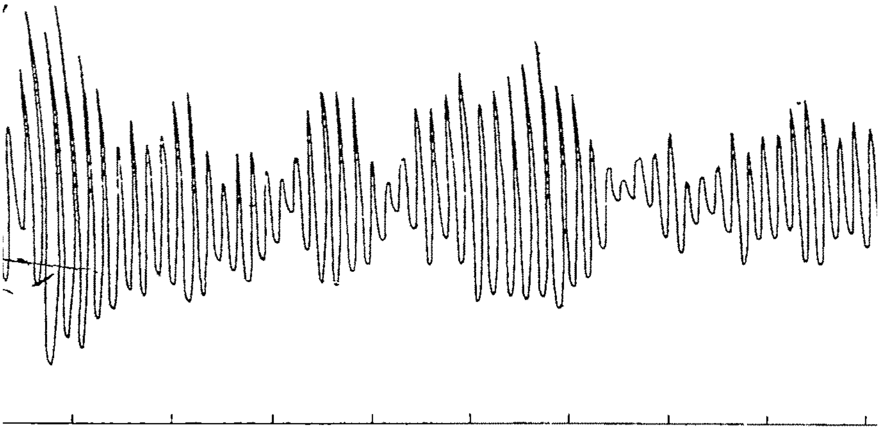
In the thalamus the internal and dorsal nuclei were alone destroyed, the ventral and lateral and the internal capsule apparently remaining intact. The greatest amount of destruction was in the mid-brain, where practically nothing remained of the tegmentum and tactal region. It seemed as though the tumour lay in the aqueduct of Sylvius and destroyed the surrounding tissues by expanding, but the pes pedunculi, though flattened out, appeared unaffected. Thus it destroyed the dorso-caudal part of each thalamus, the tectum and tegmentum of the mid-brain, including superior cerebellar peduncles and red nuclei, which were unrecognisable, and posteriorly involved the left lateral lobe of the cerebellum and the adjacent portion of the vermis.

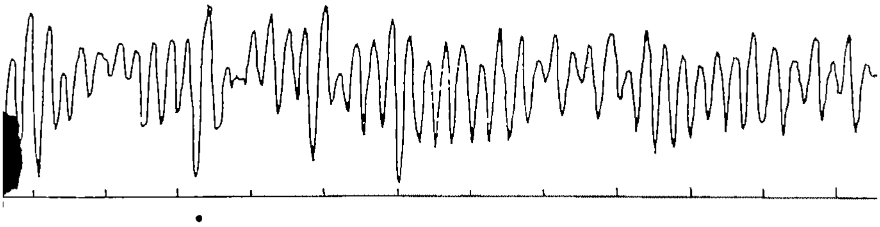
The pons varolii, medulla oblongata and spinal cord were investigated by Marchi's method. At the level of the lower part of the pons there were very few degenerated fibres in the pyramids, but considerable degeneration marked the position of the rubro-spinal tracts. Sparse, scattered black spots indicated a slight degree of diffuse degeneration, probably pressure effect.

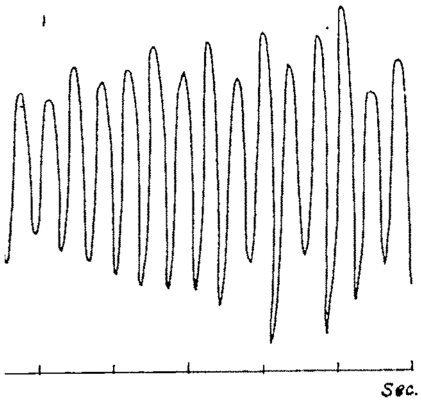
In the medulla there was only degeneration in the position of the rubro-spinal tracts, and in the cord some degeneration of large fibres, probably of the same origin, was visible in the lateral columns, and, in addition, degeneration in the dorsal columns of the type frequently met with in cases of cerebral tumour.

Case 4.—J. R., a boy of 6 years, was admitted under the care of Dr. Ferrier, in September, 1903. There had been gradually progressing weakness of the right limbs of three months' duration, with consequent difficulty in walking and reluctance to use the right hand. No facial weakness or





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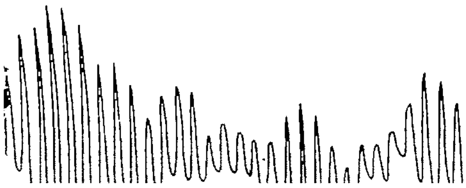

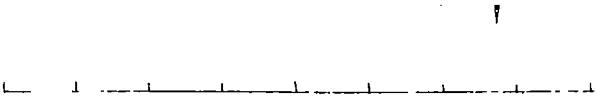

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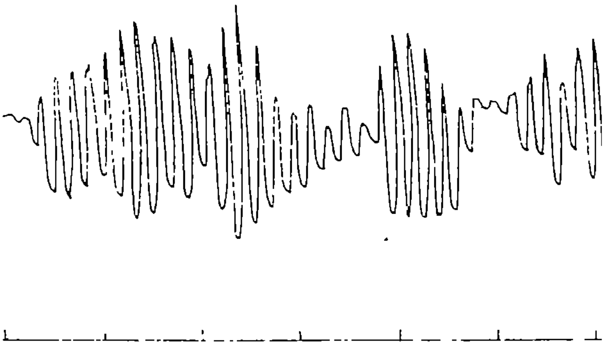

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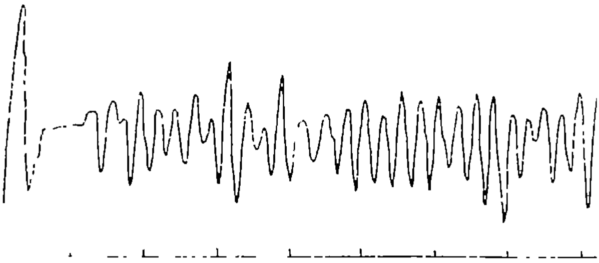

Nº3

abnormality was observed, but a squint was noticed six weeks previous to admission.

Since the weakness of the right arm became marked his mother had noticed that it was very unsteady and tremulous in voluntary movement, and that it occasionally would shake even when at rest. He had suffered much with headache, generally referred to the back of the head, and frequently vomited during the course of the illness. There had not been any affection of speech and no fits.

He was found to be a strong and healthy-looking child and bright and intelligent. His sight and visual fields were good, but intense optic neuritis was present. His head was constantly held inclined to the right and rotated to the left, so that the right occiput approached the right shoulder, while his eyes were generally in conjugate deviation to the left. It was very difficult to make him look to the right, and in so doing some weakness of the right external rectus became visible, and apparently diplopia resulted, as he tried to cover one eye with his hand. Movement of both eyes to the left was fully possible, and the vertical movements and convergence were also good, but there was coarse nystagmus on looking to the left. The pupil reflexes were normal. The right side of the face scarcely moved so fully as the left on voluntary effort, and there was some relative weakness of the right limbs but without any rigidity. All movements of the right arm were very irregular and ataxic, and some coarse and rapid tremor affecting one or any group of muscles appeared when the limb was handled or when he was asked to perform any strong movement with the left arm. The tremor was rapid: from tracings the rate seemed to be about 4 or 5 oscillations per second, and while it was limited to one group of muscles regular in range.

The distal and proximal muscles were about equally affected. The child was unable to check it voluntarily, and it was always much increased by any excitement or agitation, as when he wept. There was a slight degree of right hemianæsthesia, but the deep reflexes were about equal on the two sides, though a typical Babinski's sign was obtained on the right.

The child became drowsier while under observation, suffered considerably with headache and vomited almost daily. Within a fortnight the upward movements of both eyes were lost, and the pupillary light reaction became very sluggish, and a few days later could no longer be obtained. The right limbs also became weaker, and the above-described tremor was more frequently present. About a month after admission it began to affect the right lower extremity. As in the arm, it always appeared or was increased when the boy was agitated, or when he moved the left limbs against resistance. The right-sided hemianæsthesia also became much more definite.

Later, the downward movements of the eyes became deficient, the upward movements completely lost, and he was no longer able to converge. The pupils remained inactive to light, and the contraction on accommodation became sluggish. The weakness of the right side of the face on voluntary movement became more marked, yet this side seemed to move the more on smiling. As the paresis of the right limbs increased, the tremor began to disappear, and finally was only to be observed when the child was very agitated, as when he sobbed or cried.

The child became weaker and more drowsy, and died about two months after admission to hospital.

Diagnosis.—The right-sided hemiplegia and hemianæsthesia, with the above-mentioned ocular symptoms, lead to the diagnosis of a lesion of the left side of the mid-brain being made, and from the general symptoms, the headache, vomiting and optic neuritis, this lesion was evidently a neoplasm.

The diagnosis was fully confirmed in the autopsy, for a small tumour, which on microscopical examination was seen to be gliomatous, was found in the left side of the mid-brain. The tumour, which was almost quite spherical and about the size of a hazel nut (1·7 cm. in cross section), lay in the lower part of the left tegmentum and largely infiltrated the left pes pedunclei at about the level of the left oculo-motor nerve, completely destroying the structures immediately dorsal to it, including the substance nigra and the nucleus ruber.

Posteriorly, dorsally, and laterally this tumour was limited by a cystic cavity which contained clear, viscid, greenish fluid. It was considerably larger in all dimensions than the tumour, and caused marked displacement of the neighbouring structures. Its effect extended posteriorly into the tegmentum of the pons and frontalwards to the posterior part of the thalamus, without, however, invading either. It practically destroyed the left corpora quadrigemina, and to a great extent also the right, but its chief effect on the various structures was merely displacement. The pons varolii, medulla oblongata and spinal cord were examined by Marchi's method, but the degeneration present was limited to the pyramidal tract and Monakow's bundle (tractus rubrospinalis) on the right side, and was relatively slight in the former. The latter followed the typical course described by Collier and

Buzzard. The amount of secondary degeneration was, as is generally the case from gliomatous tumours, remarkably small in contrast to the size of the lesion.

Case 5.—E. W., a boy of five years of age, was admitted under the care of Dr. Ferrier in December, 1903. His health previous to this illness was good, but one brother had tubercular joint disease. He was a healthy and bright child till three months before he came under observation, when he began to vomit very frequently and to suffer severe headache. A short time later it was noticed that the child was reluctant to use the right arm and limped on the right leg, and about two months from the commencement of his illness the right arm began to shake and tremble when he moved it, when it lay in any "uncomfortable position," or even at apparently perfect rest when the child was excited or agitated. This tremor, or unsteadiness of the arm, slowly increased till a few days prior to his admission to hospital. Then one day he fell down unconscious, and since then has scarcely been able to move either of the right limbs.

On examination the child was found to be bright and intelligent, his visceral organs were normal, and no evidence of tubercular disease could be detected. His sight was considerably impaired, there being intense optic neuritis, and there was right hemianopia reaching almost up to the vertical line through the fixation point. Wernicke's hemianopic pupillary reaction test was positive, that is, the pupils did not contract to rays of light thrown in from the blind halves of the visual fields.

There was slight weakness of the left external rectus, and some difficulty in the conjugate movements of both eyes to the right. Though there was no visible difference between the two sides of the face while they were at rest, there was slight weakness of the right side, practically limited to its lower half, on voluntary movement. In a spontaneous smile, on the other hand, there was practically no movements of the right side of the face, though that of the left side was free, and by contrast apparently exaggerated. Some movement of the right side occurred when the child laughed aloud, or wept, but much less than of the left side. There was no paresis of the tongue or palate, and no affection of phonation or deglutition.

The constancy with which the head was held in an unnatural position was striking. It was always to be seen slightly inclined to the right and rotated to the left, so that the right side of the occiput was approximated to the right shoulder, but voluntary movement in each direction was free and strong.

The muscles of the limbs were fairly well developed, and the strength of the left pair was relatively good and their voluntary movements well co-ordinated. When, however, the child was agitated, rather fine tremor of medium rate and regular in amplitude and rate was observed in both the left arm and leg. This was never visible when the child was composed and at rest, and as it only occurred under the above-mentioned condition, and never persisted for long, its further character could not be studied. The right arm lay quite powerless and flaccid as he first came under observation, and no tremor of any part of it was observed. The right lower extremity was also very weak, but not so severely affected as the arm. He was then unable to walk alone, and with support reeled and staggered. There was no trace of aphasia associated with this right hemiplegia, even though the boy was right-handed. There was also considerable diminution of all forms of sensation on the right side of the body, the loss of the sense of position in the right limbs being relatively the greatest. All the deep reflexes were absent, stimulation of the soles of the feet gave an extensor response on the right side, but on the left flexor.

While under observation in hospital the right limbs became more rigid and there was some return of power. The voluntary movements possible, however, were complicated by an irregularity of the intention-tremor type, and spontaneous tremor was frequently observed in the right arm when the child was in any way excited or agitated, as if abruptly wakened from sleep. He apparently had no power to check or control this tremor, which, while it lasted, was of medium rapidity—about four oscillations per second, and regular in rate. It was fairly coarse, but not quite regular in amplitude. It generally resulted from forcible movements of the opposite limb, and ceased when the child was composed and the limb lay at rest.

The presence of hemianopia with Wernicke's reaction was repeatedly confirmed, till the child finally became almost quite blind, with subsidence of the optic neuritis into atrophy.

Some weakness of the upward movement of the left eye also developed while the patient was under observation.

The voluntary paresis of the right side of the face diminished, but the defect of expressional movement became more pronounced, in fact, the right side of the face was generally described as immobile on smiling.

The deep reflexes soon returned. The patient's general condition improved and the headache and vomiting ceased. The patient finally left hospital almost blind, unable to walk, but with fair range and power of movement of the right limbs.

Diagnosis.—This case so much resembles Case 4 that the same arguments serve to indicate the localisation of the lesion. The defective upward movement of the left eye, which developed while he was under observation, and the paresis of the right side of the face in expressional movements, tend to confirm the diagnosis of a lesion in the left side of the mid-brain.

Case 6.—F. H., a boy of 15 years of age, was admitted under the care of Dr. Ormerod, in October, 1903. He came of healthy parentage and has not had any serious illnesses. Two months previous to admission his left arm was first noticed to become weak, or "useless" as he described it, so that he could not employ it properly in his work, and he became unable to perform finer movements with it. This affection was from the first slowly progressive, and about a month or six weeks later the limb began to shake on movement or even during rest. For six weeks his gait had been affected, and his sight had not been so good as it formerly was. During the whole period of his illness he suffered very much with severe headache, and frequently "felt sick," though he rarely vomited.

On examination, hemianopia to the left extending up to the vertical line through the fixation point was found, and Wernicke's hemianopic pupillary reaction was present, *i.e.*, the pupils did not react to rays of light thrown into the eyes from the left side. There was no limitation or weakness of any of the ocular movements, and no paresis or loss of function of any of the other cranial nerves.

The muscles of the limbs and of the trunk were well developed for a boy of his age, but there was a very slight degree of rigidity of the left limbs. The left arm was very slightly weaker than the right, but the paresis of the left lower extremity was proportionately greater, indeed, practically no voluntary movement of the foot or toes was possible.

The most striking symptom of the case was the presence of involuntary movements, in the form of a tremor of the left limbs, but much more frequently and constantly observed in the upper than in the lower extremity. Neither the face nor tongue was ever affected by this, and it never spread to the right limbs. When the left arm was moved, or when it did not lie at perfect-rest, each segment of the limbs supporting its own weight, there was constantly coarse tremor of one or other part of it whose rate, counted from tracings, was always about 3½ oscillations per second. All groups of muscles were involved, but the distal more frequently than the proximal, and in the latter the tremor was always coarser. Flexion-extension or pronation-supination of the elbow, flexion-extension of the wrist and flexion-extension of the fingers at the metacarpo-phalangeal joints, were the most frequent movements. Though more or less rhythmical the range of the oscillations was seldom constant for long, but their rate did not tend to vary. The amplitude was increased by bringing the muscles into action, or by placing the limb in such a position that they were in a state of strain, by excitement or agitation of the patient, and by strong action of the fellow limb. It only ceased during sleep, or when the limb lay at perfect rest. Voluntary movements were complicated by ataxia and irregularity of the intention-tremor type, *i.e.*, the range of deviation from the direct line increased towards the completion of the action. Similar tremor under similar conditions was occasionally observed in the lower extremity, with a predilection to the distal joints, but the voluntary movements of the limb which were possible were fairly accurately executed. His gait was only affected by the weakness of his left limb, which, as the foot could not be fully dorsi-flexed owing to weakness, was raised high to allow the toes to clear the ground, and thrown somewhat outwards as it was advanced. There was a slight degree of left-sided hemianæsthesia, tactile sensation being the most defective. The deep reflexes were increased in the left side, and the left plantar reflex was of the extensor type. His condition has remained practically unchanged since he first came under observation, excepting that the headache and general cerebral symptoms have abated.

Diagnosis.—The left hemianopia and the presence of Wernicke's reaction indicates involvement of the right optic tract before its termination in the primary optic centres, or of the quadrigeminal region, and the left-sided hemiparesis and hemianæsthesia, associated with it, points to a lesion of the right side of the mid-brain, probably by a tumour growing from its lateral surface medianwards.

Case 7.—C. S., a woman of 37 years of age, was admitted under the care of Dr. Hughlings Jackson, in September, 1903. She complained of uselessness of the right limbs and of occasional attacks of headache and vomiting. She came of tubercular stock, and her antecedent history gave rise to the suspicion of syphilitic infection.

The affection she complained of had then lasted nearly two years, and had developed slowly after a fit, which was preceded by some vertigo. During the next week or so the right arm slowly became weaker, and about six weeks later it first became shaky and tremulous when she attempted to use it. The tremor of the right arm has slowly increased since then, and she has now for eighteen months not been able to use this limb in taking her food. She spontaneously emphasises that it is rather "uselessness" than weakness which affects this limb. She cannot pick anything up with the hand, but is able to nurse and carry her baby on this arm when she has placed him in it with the left hand.

Her gait became affected only nine months after the commencement of the illness. First the right leg felt weak and gave way under her, and soon she began to walk so unsteadily and uncertainly that she was reluctant to go out of doors lest it should be thought she was drunk. Since then she has taken very short, shuffling steps. For two months she has been troubled with difficulty in reading: the letters now run into one another and she has had double vision.

On examination, her visceral organs were found to be normal, and, though an uneducated woman, she was clear and intelligent with a good and reliable memory. She was very emotional and frequently burst into tears or laughter without adequate cause, saying she could not help it.

Her vision was good and there was no contraction of the visual fields or optic neuritis. Although she complained of diplopia there was no visible paresis of any ocular muscles, but marked nystagmus on lateral deviation, and slighter accompanying the vertical movements was present; even on fixation the eyes were unsteady. The pupils were equal and reacted well to light and on accommodation. The face seemed rather fixed and expressionless, but there was no visible paresis in voluntary movement, though in emotional expression movement was limited, slow and rather constrained. The muscles of all limbs were well developed and of good strength, with but little difference between the power of the two sides, yet it was of uselessness of the right limbs that she most complained. In the right, however, there was marked tremor of a coarse and more or less irregular character, definitely rhythmical, with frequent increase of amplitude, but from numerous tracings taken it is apparently constant in rate, about four oscillations per second. (Tracing III.). This tremor ceased during sleep and when the limb lay at perfect rest in bed, but at once commenced when she was at all excited, or even when she comes under observation, when the limb is moved about either passively or actively, or when the muscles are in any way put in a strain as, for instance, when the limb hangs by her side. The tremor was not so regular in the amplitude of its oscillations as that of the preceding cases, but regular, clonic contractions of one or other group of muscles might be observed persisting for a considerable time under favourable conditions. This was not at first apparent, the movements of the limb being generally irregular owing to the simultaneous presence of several tremors affecting different groups of muscles. It was a true tremor, in that it was due to the alternate contractions of one or more muscles and their antagonists, and might affect any group in the limb, but showed a preference to the distal. Voluntary movements, but especially the finer, were complicated by an accentuating tremor of the intention-tremor type, so that she was unable to use this limb in taking her meals and could not hold one finger in apposition to her nose. Similar tremor has not been observed in the lower extremity, but its voluntary movements are jerky and irregular.

Her gait was very peculiar; she advanced with slow, shuffling steps, but it was neither ataxic nor spastic, for she neither staggered nor dragged her feet, but took very short steps as though afraid to trust her weight on the right limb, consequently advancing the left foot as quickly and as short a distance as possible and in the next step unable to move the right foot fully forwards. She could stand, however, easily on either foot, and did not reel with her feet together and her eyes closed.

There was, besides, a definite degree of right hemianæsthesia, which extended up to the middle line, and a more marked loss of the sense of position in the right limbs. The deep reflexes were slightly brisker on the right than on the left side, but both plantar reflexes were of the normal flexor type. There was no trace of aphasia or other affection of speech. The patient remained about six weeks in hospital, but is still under observation. She is so far unimproved.

Diagnosis.—Even though the cardinal symptoms—headache, vomiting, and optic neuritis—were absent, there can be little doubt that in this case the lesion is a tumour which has been of very slow growth and has been, perhaps, for some time latent. The marked hemianæsthesia, with ocular symptoms and but slight hemiparesis, makes a mid-brain lesion of the opposite side probable, and this localisation is supported by the emotional state.

Case 8.—A. B., a man of 47 years of age, had been under the observation of Dr. Risien Russell for five years in the out-patient department of the hospital. His illness had a sudden onset during sleep, six years ago. For the next three weeks he was in a dazed condition, but not unconscious, and suffered with severe headache and vomiting. From the onset he was able to move all his limbs about in bed, but noticed that the movements of the right arm and leg were weaker than they had previously been, and were, in addition, very awkward and irregular. He had no difficulty in speaking, but people were not able to understand him easily for some time. He had, however, no difficulty in swallowing. On leaving bed he found that his gait was extremely ataxic; he reeled about like a drunken man, had to learn like a child to walk again, but it rapidly improved. His right arm has remained very ataxic; he described it as "useless," recognising that he had sufficient power to move it in any way he pleased, but that he was unable to correctly regulate its movements. Besides this defect in the co-ordination of movements, he describes a trembling of the right arm which came on when he allowed it to lie in any uncomfortable position or held it up. This tremor has been most marked when he was agitated, excited, or tired. It first appeared two or three months after the beginning of his illness, and not, as the ataxia, immediately after the onset. During the first three or four months of his illness he had diplopia, especially, he thinks, on looking down, the images being separated both horizontally and vertically, and tended to cross one another. He has never had ptosis or any affection of co-ordination.

No diplopia can now be made out, but, on looking down, the left eye moved slightly less fully than the right; otherwise his vision and ocular movements were unaffected. There was a very slight degree of voluntary paresis of the right side of the face, but the difference between the two halves was more marked in expressional movements, and as he conversed he elevated only the left eyebrow. No involuntary movements have been observed on either side of his face or in tongue. There was but little difference between the strength of the right and left limbs, the former being only slightly weaker, but there was a relatively greater degree of rigidity. The voluntary movements of both right arm and leg were very irregular, the irregularity being of the type of intention-tremor. His gait is at present almost quite natural, but he says that he must be careful and slow in going up and down stairs, as the right leg trembles. Besides this intention-tremor, there was some tremor of the right arm when he held it extended in front of him or kept it in such a position that each part did not bear its own weight. The movements may occur at any joint, but more frequently at the distal than at the proximal. It only remained a short time limited to any one group of muscles, but while it persisted was regular in rate and in amplitude, of medium rapidity, about four oscillations per second, and fairly coarse in range. He was unable to voluntarily inhibit it for any time, and on movement the intention-tremor above described appeared.

It was increased by any excitement or agitation, and constantly came on when he attempted to perform forcible actions with the left arm. It ceases when he is at perfect rest.

No affection of any form of sensation could be determined, and the reflexes were practically equal in the two sides. His gait presented no abnormal features.

There was no evidence of vascular disease, but the patient had syphilis in youth.

Diagnosis.—This rests mainly in the definite history of diplopia with vertical separation of the images, and the slight defect of downward movement of the left eye which is still apparent. These indicate a mid-brain lesion affecting part of the left oculomotor nucleus or nerve. The sudden onset of the condition suggests that it was of vascular origin.

Case 9.—J. L., a man of 57 years of age, came under the care of Dr. Risien Russell two weeks after the onset of his illness. According to his history he retired to bed one night as well as usual, but woke up at an early hour conscious that something had happened to him. On attempting to leave bed he found he was unable to stand, and though able while lying down to move his legs about, they seemed too weak, and especially the left, to bear his weight; in his own words, he "seemed to have lost control over them." He had also tingling and numbness of all four extremities, subjectively more intense on the right than on the left side of the body. There was also when he moved severe vertigo, with translation of the objects he looked at from left to right, but there was no subjective or objective auditory disturbance. When brought a drink he found he was unable to bring it to his lips, as he had also lost control of his hands, they were very unsteady, and he was unable to direct their movements accurately. His articulation was also at the time so much affected that his speech was unintelligible. He felt very ill, had severe headache and vomited persistently for the first twenty-four hours.

The more acute symptoms disappeared after the first few days, and in the course of a week or so his right limbs were again normal, his speech was quite easily intelligible, though not natural, the vertigo had ceased, and he had no longer any paræsthesia in his limbs. The weakness and uselessness of the left limbs, however, has persisted, the arm is so unsteady that he cannot employ it in any work, and the leg so weak that he is afraid to trust his weight on it when walking.

From the onset of his illness till he came under observation he was troubled with diplopia, especially on looking down or in front as he lay in bed. He gave no history of involuntary movements of the limb, except the irregular excursions associated with voluntary movement.

On examination he was found to be a healthy-looking, well-preserved man for his years, but his arteries were tortuous and rigid, and he admitted syphilitic infection some years previously. His special senses were unaffected, but on investigation of the ocular movements, there was found to be some defect in the downward movement of the right eye and diplopia, indicating weakness of the right inferior rectus. There was no affection of the functions of the other cranial nerves.

The musculature of the limbs was fairly developed, and there was practically no difference between the strength of the right and left limbs, nor was there any rigidity on either side. All movements of both left arm and leg were, however, very ataxic, the inco-ordination closely resembling that due to unilateral cerebellar lesions, and this was equally marked in bringing his finger to his nose and in touching a foreign object. There was also a very slight degree of diadokokinesia in the left arm. His gait, which, from his own account, resembled that of a drunken man during the first fortnight of his illness, was no longer ataxic when he came under observation, but he walked with short, uncertain steps, holding the left leg too stiff and bringing the foot too forcibly to the floor. For the first few days of his stay in hospital there was slight left hemianæsthesia, but it quickly disappeared. The left deep reflexes were brisker than the right, and the left plantar reflex was of indefinite character.

His general condition, and especially his gait, improved after he came under observation; but about six weeks after the commencement of his illness a new symptom developed. This was tremor of the left upper extremity, which at first was only very occasionally to be observed, but became more frequent. It showed predilection to affection of the more proximal groups of muscles, as adduction-abduction of the shoulder, with occasional rotation, flexion-extension of the elbow; but flexion-extension of the wrist and cigarette-rolling movements of the fingers and thumb were often observed. It only came out after some movement of the limb, if this lay in a constrained position, if the patient were at all agitated, or when he moved the right arm forcibly against resistance. It was fairly rhythmical in character, slow (about 3½-4 oscillations per second), and regular in rate, with some tendency to slow accentuation and decline of amplitude, which was otherwise regular. It never persisted for long. The patient was unable to voluntarily arrest it, and it increased on any excitement or exertion. While earlier the defect of co-ordination of the movements of the left arm was of the cerebellar type, it slowly became identical with the intention-tremor of disseminated sclerosis, so that on trying to bring a finger to the tip of his nose it would strike the nose repeatedly in the vain attempt to keep it in position.

Diagnosis.—The existence of a partial palsy of the right oculomotor nerve, with contra-lateral limb symptoms, indicates disease of the right side of the mid-brain. The sudden onset makes a vascular lesion probable.

In this series of cases, dissimilar though they be in their general appearance and in many of their clinical characters, there is one constant symptom. This symptom is tremor, which, it will be seen from the clinical and anatomical descriptions given above, has been observed in two classes of cases, in chronic and unprogressive cases in which the lesion is probably either of vascular origin as in Cases 1, 2, 8, 9, or, for the time being, latent tumour, as in Nos. 5, 6 and 7; and in rapidly progressive and early fatal cases of intracranial neoplasm, as Cases 3 and 4. In spite of this the tremor in all cases is very similar, or at least has constantly such characteristics as will allow of it being regarded as a definite symptom and clinical entity.

In recapitulating from the history of the individual cases, it may be described as consisting in a series of involuntary oscillations of any part of a limb, due to the alternate contractions of one group of muscles and its antagonists, of slow rate, varying in rapidity from 3 to 5 oscillations per second, in all cases more or less regular in rate while limited to any one group of muscles, in some cases (1, 3, 4) absolutely so; generally coarse, *i.e.*, of large amplitude, and either quite regular in the range of amplitude, as 1, 3, 4; with a periodical rhythmical increase and decrease of the range, as 8 and 9; or irregular, as Cases 6 and 7.

In some instances there was a remarkable tendency for the tremor to persist, apparently indefinitely, limited to the one movement if the position of the limb and other conditions remained unaltered; while in other cases it would involve various groups of muscles for short periods in succession or even simultaneously, the resulting movement of the limb being in the latter case compound and irregular, as the various oscillations were not synchronous in rate or equal in range.

In every case it was found that the patient was unable to voluntarily check the movement for more than the shortest space of time, and often the attempt to inhibit it only resulted in increase or accentuation of the range. In addition to the tremor proper, which may be described as static, as it occurred independently of voluntary or reflex movement of the part, volitional movement of the limb affected was in every case complicated by an irregularity of the intention-tremor type as met with in disseminated sclerosis, that is, the range of deviation from the direct line of the movement increased as the completion of the act purposed was approached.

In no case did it persist during sleep, though in Case 3 the patient was, during the greater part of the time that it was observed, in a semi-comatose state. It also ceased when the limb involved lay at complete rest, so supported that each of its segments was individually supported. In each case the influence of gravity on its production and existence was emphasised; any part of the limb allowed to hang unsupported was in some cases invariably, in all cases frequently, affected by tremor. This would seem to point to a certain condition of tone of the muscles being essential to, or at least concerned in, its pathogenesis, and opens up an interesting field for discussion and study of the relation of the two conditions. This, however, is avoided here with the excuse that we are still too ignorant of the full nervous mechanism concerned in the production and maintenance of muscle tone to profitably allow it.

In each case, too, it was observed that the psychical state exerted considerable influence on the intensity and character of the tremor; it always increased with any agitation or excitement of the patient, and diminished as the patient again became composed and calm. Another factor which increased the tremor was forcible movements of the opposite limb, and even the more finely co-ordinated purposeful ones. It is noteworthy that in every case it only developed some weeks after the initial onset of the illness.

Each case of this series presents other symptoms worthy of more special notice. These may be roughly, though not perhaps accurately, grouped as those associated with the symptom-complex, but not invariably present, as rigidity, paresis and uselessness of the limbs, affection of co-ordination and an emotional state; and those merely casually related or coincident, as marked loss of voluntary power, sensory disturbance, the change in the reflexes, and the affection of the ocular movements, or of the functions of other cranial nerves. At present it is only the former group which will be dealt with, as the members of the latter are chiefly of interest in determining the anatomical site of the lesions.

The rigidity presented no special character. That which is described as belonging to the symptom-complex was, in all probability, not due, as the rigidity of the usual spastic states, to involvement of the cortico-spinal tracts in the lesion, for neither the condition of the reflexes nor the general character of the limbs warranted this conclusion. Unlike the late rigidity of hemiplegia and other spastic states, it was never associated with organic contractures. It was slight in degree, and is perhaps better described as a stiffness and a constant resistance experienced in the passive movement of the limb through the whole range of the movement. This resistance was not more marked at the commencement of the movement, and remained constant through it, and in these and other particulars was dissimilar from the rigidity of spastic paralysis. Associated with it was often a marked slowness in performing, and especially in initiating, voluntary movement.

Though the patients complained of weakness and "uselessness" of the affected limbs, there was in several of the cases no paresis or scarcely any in contrast to the power of the normal limbs, that accompanying the onset of the condition in the vascular cases rapidly passing off and leaving no definite evidence of affection of the cortico-spinal tracts. This weakness of which they complained was then, in great part, merely subjective. In some cases it may have been due to the accompanying sensory loss, but where this explanation does not serve, as will be more fully discussed below, it is probably to be largely attributed to interference with other systems than the cortico-spinal. Being largely subjective it is perhaps best described by using the term employed by so many of the patients—"uselessness.' Further, it is to be noted that in all probability it was not entirely due to the affection of the co-ordination of the volitional movements.

The emotional state of some of the cases also deserves some attention. This was particularly marked in Cases 1, 2 and 7, and to a lesser extent in Cases 8 and 9. These patients all remarked spontaneously or to leading questions that their temperament had changed, and laid special emphasis on a troublesome tendency to burst into uncontrollable attacks of laughing or of tears without an adequate cause, and often quite irrelevantly to the situation. Indeed, this symptom appeared so frequently that it came to be of the greatest annoyance to the patients themselves and to their neighbours. Case 1 was often continually in laughter while being examined, and Case 7 was frequently found in tears, yet both on questioning denied any knowledge of the cause or reason of their states, and might assert that their frame of mind was in quite the reverse condition. The first three cases cited, however, occasionally complained of feeling very depressed and sad, admittedly without reason. On conversation they were found to be unnaturally dull and apathetic, a change which was also observed by their friends. They took little interest in their surroundings and exhibited a marked disinclination to exert themselves in any way, and they generally preferred to remain in bed than to mix with the other patients of their wards. There was also apparent a slowness in replying to questions or carrying out any command, a tardiness in initiating thought and action. With this psychical state some of the patients, Cases 1, 2, 4, 5, 7 and 8, exhibited a corresponding corporeal symptom. This was marked, or at least definite, paresis of the expressional or emotional movements of the face while its voluntary movements were intact or only relatively slightly affected. In Cases 1, 2, 7 and 8 the face remained remarkably placid and mask-like during conversation, with scarcely a change of expression or play of emotion, except when the patients laughed or wept, and then the movements were slow, stiff and imperfectly regulated. The affection in these cases was bilateral. In Cases 4 and 5, however, it was limited to the one side of the face, that corresponding to the limbs affected, and it was by force of contrast remarkably apparent. As the children's interest was engaged or when they smiled it was seen that the movement was limited to one side of the face, and yet on voluntary effort there was little or no paresis visible. The mental state described above was not present in these two cases.

The symptoms which may be regarded as merely coincident with the symptom-complex described, are chiefly of interest as indications of the localisation of the lesions in the various cases. They deserve to be briefly discussed individually.

While, as above-mentioned, the weakness and "uselessness" of the limbs of which the patients complained were more subjective than objective in several of the cases, in others there was marked motor paresis in the form of hemiplegia. Cases 4, 5 and 6 resembled in this respect ordinary hemiplegia of considerable severity, in Case 2 there was only slight relative weakness, with an extensor plantar response when she first came under observation, and in Case 3 the only definite evidence of involvement of the corticospinal tracts was Babinski's sign during the last few days of life, though examination of the cord failed to reveal any degeneration of the pyramidal tracts, they having probably become affected at too recent a date for secondary degeneration to have taken place. Thus, from the descriptions of the cases, it may be seen there are two varieties of weakness, that with extensor plantar response and the other signs of organic hemiplegia, and a variety, always much less intense, without accompanying signs of organic spastic paraplegia save the rigidity above discussed.

The strange character of the gait in Cases 1, 2, and 7 was very striking. Though there was little if any weakness of either limb, each of these women was reluctant to walk alone without some support or assistance, and even with it took short, shuffling steps, advancing each foot but a little distance. Each, too, kept her body bent forwards like a prematurely old person, and held it very rigid as she walked, with the arms to her sides and not swinging with each step. In none of these cases was there any reeling or staggering. It was frequently remarked how closely the gait in each of these cases resembled that of cases of paralysis agitans. The gait of Cases 3, 4, 5 and 8 was, on the other hand, very irregular and ataxic, the patients staggered from side to side, always more to the side of the affected limbs, and reeled in the drunken way characteristic of cerebellar disease. The gait of Case 7 was only affected by the weakness of the left lower extremity, and in Case 8 it was normal when he came under observation, though he stated that for some time after the onset of his illness he reeled like a drunken man.

The hemianæsthesia, permanent or temporary, observed in six of the nine cases, was always slight in degree. In no case was there absolute loss of even light touches (with cotton-wool), but the localisation of the touches was defective, and the patient was unable to feel them so distinctly as on the normal side. The diminution of pain and temperature senses was relatively equal to the tactile loss, and as it was more pronounced on the distal portion of the extremities than proximally or on the trunk, the sense of position of the affected limbs and the recognition of the shape and form of objects placed in contact with them, were only slightly defective. Despite this relatively slight degree of sensory loss, many of the patients complained of a numbness or loss of feeling in the affected limbs, that is, of subjective loss without a corresponding degree of objective change. The condition of the reflexes in the various cases depended on whether the pyramidal system was involved in the lesion, or remained intact. In the latter case they were normal, or only a slight relative increase of the knee-jerks could be made out.

Special attention must be directed to the ocular symptoms, as they form the chief aid to the localisation of the lesions. From the notes on the cases it is seen that all excepting No. 6 presented some defect of the ocular movements. Six of the other remaining eight cases exhibited weakness or loss of the vertical movements of the eyes—bilateral in Cases 1 and 4, unilateral in Cases 2, 5, 8 and 9. In each instance the upward movements were more affected than the downward, and the weakness was, where any difference between the two sides existed, greater in degree on the side opposite to that of the limbs affected. In Cases 1, 2 and 9 the lateral movements of the eye-balls remained intact. In Case 1 the complete loss of vertical movements while the lateral were perfect was remarkable, and in this respect was the complement of the condition met with in lower pontine destructive lesions. As in Case 8, the history of diplopia with vertical separation of the images was the only indication of a previous ocular palsy, it cannot be more fully discussed. In Cases 4 and 5 there was some weakness of the external rectus of the same side. In Case 3 palsy of both external recti was present when the patient first came under observation, and he later developed complete bilateral ophthalmoplegia. In Case 1 there was persistent absence of the pupillary reaction, and in Case 4 the reflex contraction both to light and on accommodation disappeared. In Case 1, too, there was defect in the power of accommodation of both eyes, and in Case 2 on the side opposite to the limb affection.

In Case 7 no definite ocular palsy could be made out, but there was persistent violent nystagmus on fixing as well as with lateral and vertical movements.

Homonymous hemianopia to the side of the affected limbs, with absence of the hemianopia pupillary reaction (Wernicke's test) was present in Cases 5 and 7.

From this discussion of the symptomatology of the cases described it is necessary to turn to the consideration of the site and nature of the pathological lesions on which the symptoms are dependent. From the general similarity of these cases and the presence of a more or less identical but exceptional symptom in all, it may be assumed that the site of the disease may be practically the same in all, and, further, it may be seen that the symptoms of each case can most naturally be explained from disease of a common region in the whole series. It is proposed to argue to the site of the disease from the symptoms independently of the *post-mortem* findings in two of the cases, and only use these latter as confirmatory of the conclusions from clinical facts.

In the first place, there is in the presence of ocular palsies in a large proportion of the cases a definite indication of the site of the lesions. This palsy most frequently took the form of loss or defect of the vertical movements of the eyeballs, certainly due to disturbance or loss of the function of part of the oculo-motor nuclei or of fibres which spring from them. The fact that there was generally only paresis of the certain muscles and not indiscriminate ophthalmoplegia, points to the affection being nuclear and not a lesion of the oculo-motor nerve trunk. That there is a segmentation of the oculo-motor nuclei and probably representation of the separate ocular muscles in its various parts, has been for long recognised, but despite the work of Hensen and Völkers, Kahler and Pick, Starr, Bernheimer, Perlia, Edinger, and others, our knowledge of this representation is far from being final or complete. The view which has been hitherto most generally accepted as being at least probably approximate to the truth is that of Starr, according to whom each oculo-motor nucleus is subdivisible into segments, arranged in a lateral and medial group, in which the ocular muscles are represented from before backwards in the following order:—

Medial.	*Lateral.*
Ciliary muscle.	Sphincter iridis.
Rectus inferior.	Levator palpebræ.
Rectus internus.	Rectus superior.
	Obliquus inferior.

This result was obtained chiefly by the compilation of clinical observations. The conclusions of other authors, chiefly from experimental work, are very divergent, probably as their results were obtained from experiments on various classes of animals. Schwabe, for instance, by Nissl's method of retrograde tigrolysis, has in the rabbit localised the two elevators of the eyes, the rectus superior and obliquus inferior, in the most caudal part of the nucleus; while Bernheimer, employing the same method in the ape, came to the conclusion that the arrangement of the central representation of the muscles from before backwards is levator palpebræ, rectus superior, obliquus inferior, rectus inferior and trochlearis, with the rectus internus and the intrinsic muscles of the globes supplied by the medial group of cells. The divergence of these views is so great that none can be applied to man without further evidence and confirmation than we now have. Bernheimer's results, as obtained in the ape, would, *a priori*, be the most probably correct, and clinical observations, chiefly personal, tend to support the representation of the elevators of the eye-balls, rectus superior and obliquus inferior, most anteriorly, and the fact that isolated lesions may paralyse the depressors together, and they alone, points to the representation of the rectus inferior in the caudal end of the oculo-motor nucleus in the proximity of the trochlear nucleus. But, as in my Case 1, the vertical movements were at one time quite lost, though the lateral were unaffected, it is difficult to accept Bernheimer's localisation of the cells of the rectus internus, intermediate between those of the elevators and depressors, especially as the lesion must have been bilateral. Clinical observations make it probable that the centre of the elevators lies close to that of the pupillary reflex arc in man, as paralysis of the upward movements was frequently associated with inactive pupils.

In this discussion it is of course assumed that there is no lower co-ordinating centre for the vertical movements of the eyes as there is for the lateral. Of this there is, as far as my knowledge extends, no direct evidence, though from analogy the existence of such seems probable.

From this consideration of the question, it must be concluded that we are not yet in a position to deduce from paresis of any one ocular muscle a lesion in any definite position of the oculo-motor nucleus, owing to the incompleteness of our knowledge.

The affection of accommodation and the pupillary reflexes is explicable, whatever be the level of representation of the vertical movements, as it seems probable that the intrinsic ocular muscles are supplied from the medial group of cells of the oculo-motor nuclei, though perhaps indirectly through the ciliary ganglia (Bernheimer, Bach, Marina and others).

It is unsafe to assume a destructive lesion of the abducens nucleus or nerve from slight palsy of the external rectus in cases of tumour, as from its long course the nerve is very liable to suffer from indirect pressure effects, as in Case 5, but the bilateral external rectus palsy in Case 3 was evidently a direct result of the lesion, the tumour extending into the fourth ventricle and compressing its floor. With the defect of the conjugate lateral movements, as in Cases 4 and 5, it is different. This, it is known, may result from a lesion of the lower co-ordinating centre of the movement—Deiter's nucleus, or of the fibres which leave it, dorsal longitudinal bundle, or, secondly, of the system of cortical origin which bears the volitional impulses for the movement. Monakow represents this tract as passing through the anterior part of the mid-brain in the formatio reticularis ventro-lateral to the aqueduct, lateral to the oculo-motor nucleus and at about the same level as it. In Case 4 it was evidently, from the autopsy, to affection of this system above its decussation that the defect was due, and the same is probably true in Case 5, as there was no clinical indication of a lower pontine lesion.

The hemianopia present in Cases 5 and 6 allows an accurate localisation of the lesion, as the presence of Wernicke's sign, inactivity of the pupil to rays of light thrown in from the blind portions of the visual field, makes it necessary to postulate a lesion of the one optic tract on the base of the brain, or at its termination in the quadrigeminal region in the primary optic centres. In each case, however, the associated symptoms pointed to the latter site, and makes it very probable that the tumour was situated in the anterior tectal portion of the mid-brain.

Another symptom in some of the cases, the localisation of whose cause is of interest, was the defect of expressional or emotional movement of one or both sides of the face without a corresponding degree of volitional paresis. It is a symptom first described by Nothnagel and since extensively studied of Bechterew, Bruns and others, and has been by all ascribed to disease of the optic thalamus, probably in its caudal end, or of an efferent tract from it to the facial nucleus. The course of this thalamo-facial connection has not yet been described, but it seems probable that it passes through the mid-brain near the raphe, as the tract of each side must lie close together to be affected by such presumably small lesions, as in Cases 1, 2 and 7. Borst, on the other hand, thinks it probable that this psycho-reflex facial tract passes through the lateral part of the tegmentum, but he does not offer any definite positive evidence in support of his view. The bilaterality in these three cases makes it probable, apart from the other symptoms, that the disease does not lie in the thalamus, but interrupts the connection of the latter with the facial nucleus, and in Cases 4 and 5, where the phenomenon was unilateral, the associated symptoms pointed to the same localisation; in Case 4 this was verified *post mortem*. The psychical state of Cases 1, 2 and 7, associated with this defect of emotional display, where there was no evidence of cortical or subcortical affection, is of interest from the psychological standpoint.

The slight degree of hemiplegia present in some of the cases could not be used as a sign in the localisation of the lesions, but its association with sensory loss, evidently not of the cortical type, points to the disease being situated below the basal ganglia, where motor and sensory tracts run in close proximity.

From these symptoms the conclusion may be drawn that the site of the lesion in each case, in some with as absolute certainty as we can attain in clinical medicine, in others less definitely indicated, is in the mid-brain, possibly in some cases extending into the posterior part of the optic thalamus.

Further, the relative escape of the cortico-spinal system indicates that the disease is limited more or less sharply, in several of the cases, to the dorsal or tegmental portion of the mid-brain.

In some of the cases (1, 2, 8 and 9) the ocular palsies indicated that it was evidently of small extent and lay close beneath the aqueduct of Sylvius, at a level corresponding to a circumscribed part of the oculo-motor nuclei. When hemiplegia of any degree and ocular palsies were together present they comprised Weber's syndrome or hemiplegia alternans superior, the cranial nerve symptom being on the side opposite to the hemiplegia. In other cases instead of ocular palsies there was homonymous hemianopia, indicating involvement of the anterior end of the mid-brain and a more lateral position of the lesion. The autopsies in Cases 3 and 4 confirmed this diagnosis and localisation, as in the latter case the tumour was limited to the region indicated, and in the former its chief mechanical and destructive effect was exerted at the same level. In Case 7 there is less definite evidence of the site of the disease, but the persistent nystagmus and the history of diplopia make it at least probable that the same region was affected as in the other cases.

In studying the pathogenesis of the tremor, the symptom common to the series of cases in which our interest is at present centred, it is necessary to determine as far as possible the essential and constant factor in the lesions concerned. This may offer considerable difficulty, for the mid-brain, besides being as it were a bridge between the fore-brain and the lower receptive and effective centres and the structure through which the impulses afferent and efferent which functionate in our ordinary existence pass, contains certain structures of its own, and sends efferent tracts to the cord, while some of its nuclei are links in the systems connecting the higher and lower centres.

But if this tremor is regarded as a symptom peculiar to or dependent on a lesion of this region, as seems probable from its association in the above series of cases and in several others which have been collected from literature, it is necessary to connect or correlate it with affection of some one portion of this part of the brain. The conclusion has already been drawn that in each of the series of cases here presented a focus of disease occupied or involved the dorsal or tegmental portion of the mid-brain. Of the structures contained in it to whose involvement the tremor may be ascribed, it is natural to first come to the consideration of the large nucleus which occupies its ventral and mesial part, and the ascending and descending systems in connection with it. This is the nucleus ruber, whose structure and anatomical relations and connections have become fairly accurately known of recent years. Developmentally it is a portion of the thalamencephalon, and its anterior end projects within the usual anatomical limits of the thalamus, while caudally it extends so far into the mid-brain that the roots of the oculo-motor nerves pass through or in close relation to it. As it thus lies ventral to the oculo-motor nuclei, and in close relation to the roots of the nerve, it seems probable, from the frequency of ocular palsies in the series of cases, that it must have suffered in the lesions, and that its damage may be the essential factor in the causation of the tremor. As is known the greater bulk of the superior peduncles of the cerebellum end in or around it, their fibres being in great part a system efferent from the cerebellum, and as they decussate before they reach it its relation to the lateral lobes of the cerebellum is crossed.

Any direct connection of the superior cerebellar peduncles with the fore-brain cortex may, with considerable certainty, be excluded, though Monakow believes that there is an indirect relation through the gray matter of the tegmentum, and possibly, too, through that of the basal ganglia. The nucleus ruber, according to the same author and others, has, in relation to its size and in comparison with the other nuclei of the thalamencephalon, but little connection with the cortex. Secondary change could be found in it only after lesions of the opercular region in man, and then in the form of slight atrophy of the ground substance while its cells remained normal, so the connection must be efferent from the cortex. On the other hand, each nucleus ruber sends fibres caudalwards to the cord—tractus rubro-spinalis (Monakow's bundle), and as these decussate almost immediately after their origin, each nucleus is in connection with the contra-lateral half of the cord. From these facts each nucleus ruber stands in relation to the opposite lobe of the cerebellum, the opposite side of the cord and to the homolateral optic thalamus, and possibly to the other basal fore-brain ganglia, and through them indirectly to the pallium of the same side. In this way quite apart from its cerebral connections it forms a limb in a system which connects each lateral lobe of the cerebellum with the same side of the spinal cord. This may be known as the cerebello-rubro-spinal system. From phylogenetic grounds it is presumably of considerable importance, and from its anatomical relations it cannot but be of physiological significance, as it comprises the chief efferent cerebellar system and is one of the most definite and direct routes by which the cerebellum may influence the lower motor centres as well as the fore-brain. It seems from anatomical and other considerations that it is to some involvement of this system that the tremor above described is to be attributed. The arguments in favour of this view may be stated as:—

Firstly.—The clinical symptoms of my cases make probable, and the cases which came to autopsy confirmed, an involvement of either the superior cerebellar peduncle, or of the red nucleus. Further, in a large number of cases collected from the literature, in which the localisation of the disease was confirmed by microscopical examination, either of these structures was involved. In some of these cases the lesions were more or less accurately limited to the involvement of these parts.

Then, too, in cases of destruction of the portions of the mid-brain other than that containing the cerebello-rubral system, no such tremor has been observed. This statement can easily be fully substantiated from personal observation as well as from cases published.

Secondly.—The character of the tremor and the conditions of its existence seem to be identical or closely similar to the tremor described by Ferrier and Turner, as resulting from section of the superior cerebellar peduncle of the same side in apes (Macacus.) The descriptions of the authors are, "except when quite at rest, a constant tremor was observed in the homolateral arm and leg, which passed into larger oscillations on volitional effort"; "the limbs were the subjects of fine tremors which became greatly amplified on exertion."

Strongly in favour of this view that this tremor was directly the result of lesions of the first link of the cerebello-rubral system is the fact that they were homolateral to the injury, while, when the nucleus ruber was involved, *i.e.*, above the decussation of the peduncles, as in the clinical cases cited, it was contralateral to the lesion.

Thirdly.—It seems to me probable that such tremor may result from interruption of the final link of this system, that is, of the tractus rubro-spinalis. In a brain which I had the opportunity of examining anatomically, a small tubercular focus destroyed this tract in about the middle of the pons, and it alone. No definite clinical history of this case was obtainable, as the patient, I believe, only came under observation when moribund from hæmoptysis; but he was said to have had for some time tremor of the limbs of the same side. Such an indefinite fact, however, cannot be used as serious argument in support of the view expressed, especially as nothing is known of the character of the tremor.

Of greater importance is a case which has been under the care of Dr. Ormerod in the National Hospital. He was a boy of 17 years of age, who presented unmistakable signs of a tumour in the right half of the lower part of the pons varoli, *i.e.*, left hemiplegia and hemianæsthesia, paralysis of the right external rectus, later of the left in addition, and finally of the lateral conjugate movements of the eyes; complete flaccid palsy of the right half of the face and progressive deafness on the same side. The power of the right limbs remained good, but a few days before death they presented slow rhythmical tremor similar to that in the cases above described. The autopsy verified the diagnosis, and the appearance of the tremor was explained by recent degeneration of the right rubro-spinal tract, visible by Marchi's method. Here it may be observed that as both the superior cerebellar peduncles and Monakow's bundles decussate in the neighbourhood of the red nucleus, the one before entering it, the other after leaving it, the tremor, if due to an affection of the cerebello-rubro-spinal system must be homolateral to the lesion if this be situated caudal to the decussations, as was so in the case just mentioned.

Fourthly.—If the tremor be due to a lesion of the cerebello-rubro-spinal system in any part of it, it should be associated with a homolateral lesion of the superior cerebellar peduncle or of the origin of this in the nucleus dentatus of the cerebellum, as Ferrier and Turner described it in the ape. As no such case has come under personal observation it may be permissible to cite one from the literature to complete the study. Sander has published a case in which the right limbs were constantly the seat during voluntary rest of a "coarse oscillatory movement and of intention tremor, quite unlike cerebellar inco-ordination, or voluntary action." Further, there was weakness of the left limbs, and the gait was reeling. In the autopsy a tumour was found in the right lobe of the cerebellum, compressing the same side of the pons and destroying the nucleus dentatus and superior cerebellar peduncle. Touche and others have published similar cases.

This case, then, is consistent with the hypothesis raised in this paper, and illustrates the association of tremor and disorder of co-ordination of the limbs of the one side with motor weakness of the opposite limbs.

If it be then admitted that the tremor as described is or may be due to an involvement of the nucleus ruber, or if some part of the rubro-spinal system, it next becomes necessary to consider its pathogenesis, the mode of its causation. In this attempt it is first essential to decide whether the lesions are to be regarded as negative or as positive, whether the symptom is the result of the merely destructive lesion of the part or of an irritative effect the lesion may have.

Either of these alternatives may be considered possible when the affection is the result of a tumour or growth, either progressive or for the time being latent, but in several of the cases where the pathological condition was in all probability due to a vascular lesion and the symptom persisted for long after its onset, it is extremely improbable that this continued to act as an irritative focus giving rise to positive symptoms. This being so in several of the cases it must be assumed from the similarity of the symptom in all, that it is to a destructive effect on the parts concerned that the tremor is to be attributed.

But as negative lesions cannot directly produce positive symptoms, other centres or parts of the central nervous system must be concerned in the genesis of the tremor, and a full consideration of the symptom leads us to the determination of these centres to whose abnormal activity the symptom may be directly due.

As in man the fore-brain cortex and the efferent tracts which leave it to pass to lower centres are so directly concerned in the maintenance of the normal condition of the peripheral motor system, and as this is affected by each slight modification, either structural or functional, of the cortex and its connections, it is justifiable first to consider if the latter may be, or may contain, the mechanism to whose over-activity or to the perversion of whose function the super-positive symptom of tremor is directly due.

The same argument has been accepted by various authors who have contributed to the discussion of this or of similar symptoms, and has been supported by a considerable array of facts. It has been especially emphasised by Massalongo. The probability that it is to such over-action that the physiology of the tremor is to be attributed is strengthened by certain deductions, apparently natural and logical, from the symptomatology of the cases described.

Thus in the *first place* the tremor was in every case observed to cease during relative cortical inactivity, as in sleep and during perfect rest of the part involved by it. If not to be regarded a necessary result, it is at least a natural corollary to the association of the involuntary movements with the over-action of any centre, that they should vary in degree or even bear a definite relation to the normal activity of the latter. For, in considering disease, it is necessary to assume that unnatural activity or function of any organ varies only in quantity or quality from the natural, and is not different in kind.

Secondly, the converse of this was present in each case, *i.e.*, the tremor increased with cortical activity, in psychical, as with any excitement or agitation of the patient, and in volitional, as in discharge of motor impulses. In the latter case the increase or accentuation of the symptom coincided not only with volitional discharge from the areas of central representation of the limbs affected, but also with movement of the opposite limbs, provided that it was sufficiently forcible, which may be interpreted in the result of the overflow or diffusion of normal discharges.

Thirdly, the tremor could in no case be controlled by volition, and frequently only increased with the attempt. On the other hand, were it the result of the over-action of a lower centre, it seems probable that the dominant system of our nervous organisation should have at least some power to inhibit it.

Finally, it was indicated in each case that there was no direct involvement or only relatively slight affection of the cortico-spinal tracts, and, further, as in Case 4, the tremor ceased as the hemiplegia increased. This fact in conjunction with the absence of such tremor in cases in which there has been any extensive destruction of the pyramidal system, even though the site of the lesion be the same as in the cases above recorded, makes it probable that for its appearance it is necessary that the cortico-spinal system should be relatively intact.

If it be then admitted that these tremors are due to cortical over-action, secondary to a lesion of the cerebello-rubral system, it must be assumed that, under natural conditions, the latter structure exerts an inhibitory influence in the former. Thus the final question arises as to whether this inhibitory function is normally exerted on the higher or on the lower motor centres, on the cortex or on the spinal cord; whether the cortical influence is regulated or modified at its point of origin, or inhibited and co-ordinated as it acts upon the spinal centres? There is not sufficient evidence available to definitely decide this question, but the two cases above cited, in which the rubro-spinal tract was interrupted below its origin, point to the probability of the latter alternative; but neither case is free from objection, as in the one the lesion was extensive, and in the other the clinical character of the symptom was not definitely ascertained.

If the other alternative is accepted, that is, that the inhibitory action of the cerebello-rubral system is exerted on the cortex, possibly through intermediary of the optic thalamus or other basal ganglia, the tremor should also result from a lesion of these parts or of the cerebralward continuation or connection of the superior cerebellar peduncle or nucleus ruber. As my clinical material does not include any such case, the question must be considered from any evidence to be obtained from the literature.

Apart from this, it may be finally concluded from the cases personally observed and above described, that tremor of the character found in these cases is the result of disturbance of the functional equilibrium of cerebellum and cerebrum, which is normally maintained by efferent impulses from the cerebellum, leaving it by its superior peduncle. It seems probable, too, that when its character is recognised it may be used as a clinical sign in the diagnosis and localisation of cerebral lesions. Too much importance must not, however, be attached to its absence or to variations in its character, for if it be, as it is assumed to be, due to a disturbance of a physiological equilibrium, there is much room for the play of other factors, according to the extent and nature of the lesion.

The close resemblance the symptoms of some of these cases bear to the classical symptomatology of paralysis agitans is at once evident. There is the same tremor during rest, regular or irregular in the time and space of its oscillations, slow in rate, and, as in many cases of paralysis agitans, it is intensified on the voluntary movement of the limb affected. A certain degree of paresis and marked rigidity, neither, however, due, in all probability, to disease of the cortico-spinal system, also formed part of the symptom-complex. In some of the cases there was the same mask-like expression, due to fixity rather than to obliteration of the facial lines, a similar rigidity of the whole body in progression, and a gait of short, shuffling steps, without, however, a tendency to propulsion or retrogression.

Other authors have drawn attention to this resemblance in publishing their cases. Raymond, for instance, remarks on "Un aspect général de raideur, de soudure, d'immobilité dans l'attitude qui rapelle un peu le facies de la paralysie agitante." This resemblance was so pronounced in a few of my cases that it leads to consideration of the question whether there can be any relation between the origin of the two conditions. As hitherto little has been discovered of the pathology of paralysis agitans, it is rash to go to hypotheses, even though there be so strong a similarity in their component symptoms, but it seems permissible to suggest the possibility of the pathology of paralysis agitans being some disease of the cerebello-rubral system. I would venture to suggest the possibility of its site being in the red nucleus, and that it consists in a slow and chronic sclerosis secondary to a vascular condition. In the first place, paralysis agitans appears as a rule in older patients, in whom there is often some degree of arterio-sclerotic disease, and experience in our *post-mortem* rooms teaches that is impossible to exclude such change in the cerebral vessels from absence of any evidence of it elsewhere. The larger arteries at the base of the brain, and especially the basilar, from which the blood supply of this region comes, are known to be particularly liable to it and to suffer often the most severely when it is general. Nor is it needful to postulate a local arterio-sclerosis, for, as the nucleus ruber is one of the most vascular portions of the central nervous system, it can be assumed that for its function a liberal supply of blood is necessary, and that consequently it would suffer the most in any general reduction of the same. Should this suggestion prove correct, the sudden onset and exacerbations of the symptoms in many cases would be intelligible, and their occasional association with shocks, moral or physical, or other conditions which may modify or act on the cerebral circulation.

A full review of the literature on such conditions as the above or of closely-allied states, is at the same time very difficult and more or less unpromising of profitable results. This is chiefly by reason of the fact that there has hitherto been but little attempt to distinguish between or classify the various forms of disorders of movement which have been recognised and described, and it is scarcely possible now, as in a large proportion of the published cases there is no accurate description of the clinical observations, many of the authors being contented with describing what they observed merely by a conventional term, or even in unconventional nomenclature. This is more or less pardonable, as there is admittedly nothing more difficult than to accurately convey by description movements observed, but the result is that this absence of accurate and systematic description makes it almost impossible to utilise in a scientific study the large material which has accumulated during the last thirty years As an instance of this may be mentioned the evident variety of conditions to which the name athetosis has been attached, and undoubtedly as large and dissimilar a series of cases has been included under the term chorea. The earlier authors included various forms of involuntary movement under some generic title, and subsequent observers have frequently been content to make some such title the diagnosis of their condition, without devoting themselves to a full clinical and anatomical study of their cases, and a scientific correlation of their results.

Post-hemiplegic chorea has been a favourite generic name for the several varieties of the symptom, but more preferable, as less binding, is the term post-hemiplegic disorders of movements, suggested by Gowers. Yet this term, too, as that proposed by Wier-Mitchell—post-paralytic chorea—is inexact and unsuitable in a certain proportion of the cases, as the involuntary movements may precede any paralysis, or may never be associated with such, nor does it express the spontaneous nature of the movements.

Yet there is such evident difference between choreoid, athetotic, tremulous and other varieties of involuntary movements when they are analysed, that it seems warranted to rigidly separate them in clinical study, and to assume that they must be due to, or constantly associated with, lesions different in nature or site. It will be seen that the definition of tremor I have adopted excludes the voluntary movements which characterise chorea, myoclonus, the classical athetosis of Hammond, and spasmodic movements due to cortical discharge (Jacksonian fits), as in none of these is there a close succession of alternate contractions of one group of muscles and its antagonists.

Physiologically, too, the class of involuntary movements under consideration seems to differ from these others in there being at least a low degree of co-ordination or regulation of the muscle contractions, which is not to be observed in choreic, myoclonic, or Jacksonian spasms. The mobile spasms of true athetosis may, perhaps, be regarded as to some extent co-ordinated movements, but it needs little argument to show that even if related in causation or origin, they differ so much in effect that the one form must be considered at least an essential modification of the other.

There has been much speculation as to the nature of their pathogenesis. Almost the earliest attempts were to associate their appearance with some affection of the cortico-spinal system—either positive, *i.e.*, irritative, as Kahler and Pick, or negative, as Charcot, Bidon, Kolish, and the majority of the subsequent French observers. This view was also put forward by Sharkey in the Gulstonian lectures of 1886. According to Kahler and Pick, the lesion, or some part of it, lies near the pyramidal system and directly irritates it. On the other hand, those who have regarded a destructive lesion of the pyramidal system the essential condition, the involuntary movements may be either an early expression of a progressive lesion (Sharkey); or may be the permanent result of fully-developed or retrogressive disease, which, however, has been only partial (Bidon, Charcot); or may be the direct result of the consequent lateral sclerosis, as Charcot at one time held. On the hypothesis of Rendu and Gombault, involuntary movements occur when the tracts are incompletely destroyed, or compressed in such a way as to hinder the regular and continuous transmission of motor impulses, without completely interrupting them. Owing to this resistance there is an accumulation of energy central to it which can only from time to time break through it, so that the voluntary impulses reach the lower motor centres intermittently, and not, as under normal conditions, continuously.

Similarly Eulenberg and others, and especially Massalongs, regard the involuntary movements as of purely cortical origin, and Demange has published a case which has seemingly supported this view. The later authors have recognised that neither of these theories are compatible with facts in a certain proportion of the cases, and the greater knowledge of to-day, attained by the very large number of cases which have been published, allow an emphatic refutation of them.

In the first place, in many of the cases which have been examined anatomically the lesion has not lain in the proximity of, or involved any part of, the cortico-spinal system ; then lesions which fulfil these conditions are frequent, while post-paralytic disorders of movement are relatively rare. Such symptoms, too, have been found practically only in disease of the region extending from the internal capsule to the upper margin of the pons, while, as the pyramidal tracts are identical in constitution throughout their whole length, an injury to them in the bulb or spinal cord should produce the same symptoms as elsewhere. Then in a considerable proportion of the cases there has been at no time such definite voluntary paralysis as accompanies lesions of the pyramidal system. These spontaneous movements are also unlike those which result from affection of the cortico-spinal system, and in certain cases (where the disease has been in the mid-brain or cerebellum) this symptom has been homolateral with the lesion.

One of the first attempts to connect the symptom with any circumscribed region was made by Charcot, who assumed that there is in the posterior limb of the internal capsule, in front of the sensory system, a layer of fibres with peculiar motor functions, whose disease leads to hemichorea. This view was later adopted by Raymond and Brissaud. The fact, however, that in a majority of the cases no disease has been found in this region has made Charcot's assumption of a system with such specific functions untenable. Soon after, Gowers, drawing attention to the constant absence of such involuntary movements in completely paralysed limbs, attributed their occurrence to "damage to the gray matter of the brain, to local perverted nutrition of the nerve cells, in consequence of which they overact, either spontaneously or on the stimulus of a volitional impulse, which is by their over-action perverted or irregularly distributed;" and he assumes that this gray matter must be in connection with the volitional centres. From the result of the anatomical examination of one of his cases he further asserts that this gray matter is that of the optic thalamus. In a later publication Gowers suggests that the symptoms may be due in many cases rather to the quality of the lesion than to its site. Monakow closely follows Gowers in regarding "irritative disease of the optic thalamus, in the form of multiple small foci," as the direct cause, assuming that from these abnormal impulses ascend to the motor cortex. Since then this localisation of the lesion has been widely accepted, and it has in many cases been verified by autopsy. In every case the disease has been limited to the posterior end of the thalamus, with the doubtful exception of one recorded by Lauenstein, in which hemiathetosis resulted from an affection of the anterior end. That in the majority of the cases the lesion has been unprogressive, of long duration, and often only the remains of vascular disease, as well as the permanency of the symptoms, are, however, strong arguments against the view of the pathogenesis of the symptom accepted by these authors. Benedict had still earlier assumed that the essential condition was an affection of some accessory motor organ which has a regulating influence on the primary motor system, and pointed out its association with disease of the mid-brain.

The accumulation of published cases with a number of autopsies showing very various sites of the disease, has caused the later authors to take a more comprehensive view of the subject. Muratow, for instance, regards the symptom as due to a disturbance of the equilibrium between the inhibitory action of the cerebellum and the tonic influence of the thalamus, on the spinal cord. Anton, in his earlier work, assumed a disturbance of the equilibrium between the centres for automatic and those for voluntary movement. Theoretically this might occur anywhere that the connections of the centres are relaid in gray matter, but it most frequently results from disease of the basal ganglia, and especially of the lenticular nucleus. It would thus be due to loss of the inhibition which the basal ganglia normally exert on the cortex. In a later publication he assumed the direct cause to be the destruction of a centrifugal tract of cortical origin, which passes to the cord through the cerebellum.

Bonhoeffer, on the other hand, regards the condition as the result of the interruption of a cerebro-petal tract which carries co-ordinating impulses to the cortex, probably from the cerebellum, and asserts that the relative integrity of the cortex and pyramidal system are necessary for its appearance. Only part of these co-ordinating impulses reach the cortex, the rest becomes disseminated in the basal and mid-brain ganglia, and from there passes to the cord through centrifugal tracts. In this way he explains the existence of automatic movements.

Nothnagel and others have expressed the opinion that, though it may result from disease of fibres in relation to the optic thalamus, the symptom has no localising value, and may be due to the central irradiative of volitional impulses.

I have been able to collect sixty cases, with autopsies, from the literature which during life presented the symptoms of spontaneous involuntary movements. Only those were taken for statistics of which there was a definite description of the nature of the involuntary movements, though in many cases, for the reasons above mentioned, it can scarcely be hoped that the description was either true or accurate; yet they serve at least to give an indication of the site of the disease associated with each variety. On the other hand, cases have not been included in which the lesion was either multiple or too extensive to warrant conclusions.

For the purpose of tabulation the involuntary movements have been divided into three classes, namely, athetosis, chorea, and tremor. The published records of fifteen cases of the first form were investigated. In four cases the lesion was accurately limited to the optic thalamus, in six it was chiefly situated in the basal ganglia, frequently involving the thalamus too; in two cases the internal capsule was the chief site of the disease; in one case the corona radiate, and in another the mid-brain was involved.

Movements described as hemi-chorea resulted in two cases from affection of the optic thalamus; in five cases of the other basal ganglia, but especially of the nucleus lenticularis; in ten cases from mid-brain lesions; in two from tumour of the pons and bulb, and in one from disease of the cerebellum.

Twenty-five cases were found in which, from the description, the involuntary movements seem to fall within the definition of tremor above given. In three of these cases the lesions were situated in the thalamus and basal ganglia; in twenty cases in the mid-brain; in two cases in the cerebellum; and in one in anterior part of the pons, extending dorsalwards, to compress or directly involve the cerebellum.

No trustworthy case has been published in which the lesion was limited to the cortex. In that of Demange almost one-half of the hemisphere was involved in the softening, and the deep portions of the brain were not carefully enough examined to exclude disease in them.

As regards the variety of the symptom found associated with disease of the different regions, we see that in twenty-one cases the disease was limited to the optic thalamus, basal ganglia, and internal capsule; and in eleven of these cases athetosis resulted; in seven choreiform movements; and in three tremor. In thirty-one cases the disease was in the mid-brain, and in these the relative frequency of the various forms are reversed, athetosis only appearing once, choreiform movements ten times, and tremor twenty times. In three cases the cerebellum was involved, in the one producing choreic symptoms, and tremor in the other two cases. In three cases, too, the pons varolii and medulla were affected, producing in two of the cases choreiform, and in one tremor-like movements. And, finally, in one case, in which the movements took the form of athetosis, there was some destruction of the corona radiata.

Although these figures may not accurately represent the relative frequency of the different varieties of involuntary movements, or of the sites of the lesions associated with them, it may safely be assumed that athetosis almost always results from disease of the basal ganglia, but especially of the optic thalamus, that choreiform movements occur with about equal frequency in lesions of the basal ganglia and of the mid-brain, while in the large majority of the cases of tremor the lesion involves the mid-brain.

Finally, it remains necessary to point out that these facts are entirely consistent with the conclusions arrived at from the study of the cases above recorded, viz., that tremor, associated with focal cerebral disease, may depend on a negative lesion of the mid-brain, or rather of the cerebello-rubral system. It must be remembered that the nucleus ruber is phylogenetically, as well as developmentally, a portion of the thalamencephalon, and though it is generally regarded as a mid-brain structure, it projects so far into the 'tween-brain that direct injury of it must frequently result from disease which is described as limited to the thalamus.

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THE PATHOLOGY OF INFANTILE PARALYSIS (ACUTE ANTERIOR POLIOMYELITIS).

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THE pathology of acute anterior poliomyelitis is a subject around which very considerable discussion has from time to time taken place. Briefly stated the views which are and have been held are as follows :—

(1) That the condition is due to a direct affection of the anterior horn cells.

(2) That the condition is due to an acute inflammation in the region of the anterior horns.

(3) That the condition is due to a vascular lesion which may be either thrombotic or embolic.

There is, I believe, no doubt that cases occur which belong to the first group, but they do not clinically conform to the usual type of acute infantile paralysis, but have a more gradual onset, a wider distribution, and the final condition is one of widely distributed weakness, and it may be atrophy of muscles, and not the localised weakness and atrophy seen in the more usual form of infantile palsy. It is not my intention at the present time to discuss this point.

The view that is most commonly held of the pathology of acute anterior poliomyelitis is that the condition is due to an acute inflammation which may be of bacterial origin, or may be due to some form of poison of unknown origin having an especial selective action on the anterior horn.

The third view that the condition is directly due to thrombosis or embolism is that held by but few observers, and it is with the object of showing that this is probably the most correct view that the three following cases are now recorded.

Of the three cases described below, the first died on the thirteenth day after the onset of the disease, the second died three months after the onset, and the third two years after the onset.

Case 1.—F. L. aged 14 months, was in perfect health till nine days before he was first seen; he could use his arms well and could walk with a little assistance. One morning his mother noticed that the child was feverish, and on the evening of the same day she noticed that his back was weak, and that his head fell about anyhow; during the next few days his weakness increased, so that in three days after the onset he was perfectly helpless in all his limbs, and his voice became very feeble. There was no vomiting or diarrhœa, and the child had had no preceding illness.

On admission to the hospital the child was completely paralysed in all four extremities; there was complete paralysis of the muscles of the back and neck and paralysis of the intercostal muscles. The knee-jerks were absent, but sensation was perfectly normal.

The muscles of the arms showed the reaction of degeneration, but a complete examination was not made owing to the feeble condition of the child. The temperature varied from 99° to 100° F. in the evening, falling to normal in the morning. Three days after admission the child suddenly died while being fed.

Pathological examination.—To the naked eye the cord appeared perfectly normal and all the organs of the body were healthy. The cord was placed in Muller's fluid, and, after hardening for six months, was passed through the usual media, imbedded and cut in colloidin. Sections were stained by the Weigert-Pal method and with hæmatoxylin, eosin and carmine.

On examination of the specimens stained by the Weigert-Pal method nothing obviously abnormal could be detected, the medullated fibres of the white matter were well stained, and the finer fibres of the gray matter in the posterior portion of the cord were normal. In the anterior horns, however, the fine medullated fibres were more broken up and separated than normal. Sections taken from the lumbar region which had been stained with eosin and hæmatoxylin showed very considerable inflammatory change in the gray matter of the anterior horns. The vessels were widely dilated and surrounded by numerous small round cells which lay in the perivascular lymphatic space. Many of the vessels were thrombosed, and small extravasations of blood could be seen at various points (plate I., fig. 1). The dilatation of vessels was practically limited to the ventral portion of the cord, and was far more marked in the gray matter of the anterior horn than in the white matter, but the vessels passing through the white matter were undoubtedly affected. A few nerve cells could be seen situated as a rule close to the junction of the gray and white matter. The anterior median artery and its larger branches could be seen to be thrombosed.

In the thoracic region of the cord the inflammatory process was still present, but was not so intense as in the lumbar region; the cells of Clarke's column were normal.

In the cervical region a similar process was present, but to a lesser degree, and several normal cells could be seen in the anterior horns.

Even in the medulla there was perivascular exudation, although there was very little change in the nervous structures.

The case was under the care of the late Dr. Sturges, at the Children's Hospital, in 1893. No bacteriological examination was made, nor was the cord examined by Marchi's method.

Case 2.—The second case was that of a female child, aged 6 months, who was admitted to the Hospital for Sick Children, under the care of Dr. Garrod, on October 4, 1900, and to him I am indebted for permission to publish this case.

The history of the case was that the child eight weeks previous to admission awoke screaming, and the mother found that the child was powerless in all four extremities, on the following day it cried when moved, and on the next day lost its voice, and it had not coughed or sneezed since.

After fourteen days it recovered the use of its arms, but no return of power took place in the legs. It was the first child, and up to the onset of the illness was perfectly well and had been breast fed.

On admission to the hospital the child was well nourished, it could hold its head up, but was unable to sit up; there was an internal strabismus. The chest was well developed, but the respirations were almost entirely diaphragmatic. The arms were moved quite naturally, but the legs were absolutely flaccid and powerless, the knee-jerks were absent, and the plantar gave a flexor response. There was some difficulty in swallowing, but no regurgitation of fluid through the nose and the palate moved well. The child developed broncho-pneumonia, and died some five weeks after admission.

At the autopsy a considerable amount of broncho-pneumonia was present. The brain and spinal cord appeared normal on their external surfaces. Cultures were taken from the spinal fluid and from the gray matter of the cord, and the following was the report furnished by Dr. Thursfield.

(1) Cultures from the fluid grew:—

(*A*) a short bacillus taking the stain with some difficulty and growing in great profusion. (I believe this to be proteus vulgaris.)

(*B*) A fat coccus growing very scantily, strongly resembling cocci from infected wounds.

(*C*) A large number of ordinary staphylococci.

(2) Cultures from the anterior horns grew:—

(*a*) A short bacillus taking the stain badly, but apparently not identical with 1 *A*.

(*b*) The same fat coccus above described.

There were no staphylococci, and both the above were probably ordinary contamination bacteria.

After the cord had been hardened in formalin for three days, sections were made in a transverse direction. The medulla, cervical and thoracic region appeared normal. At the level of the first lumbar segment the anterior horn appeared redder than normal, and in all the lumbar and upper sacral segments both anterior horns were red and softened and had a gelatinous appearance.

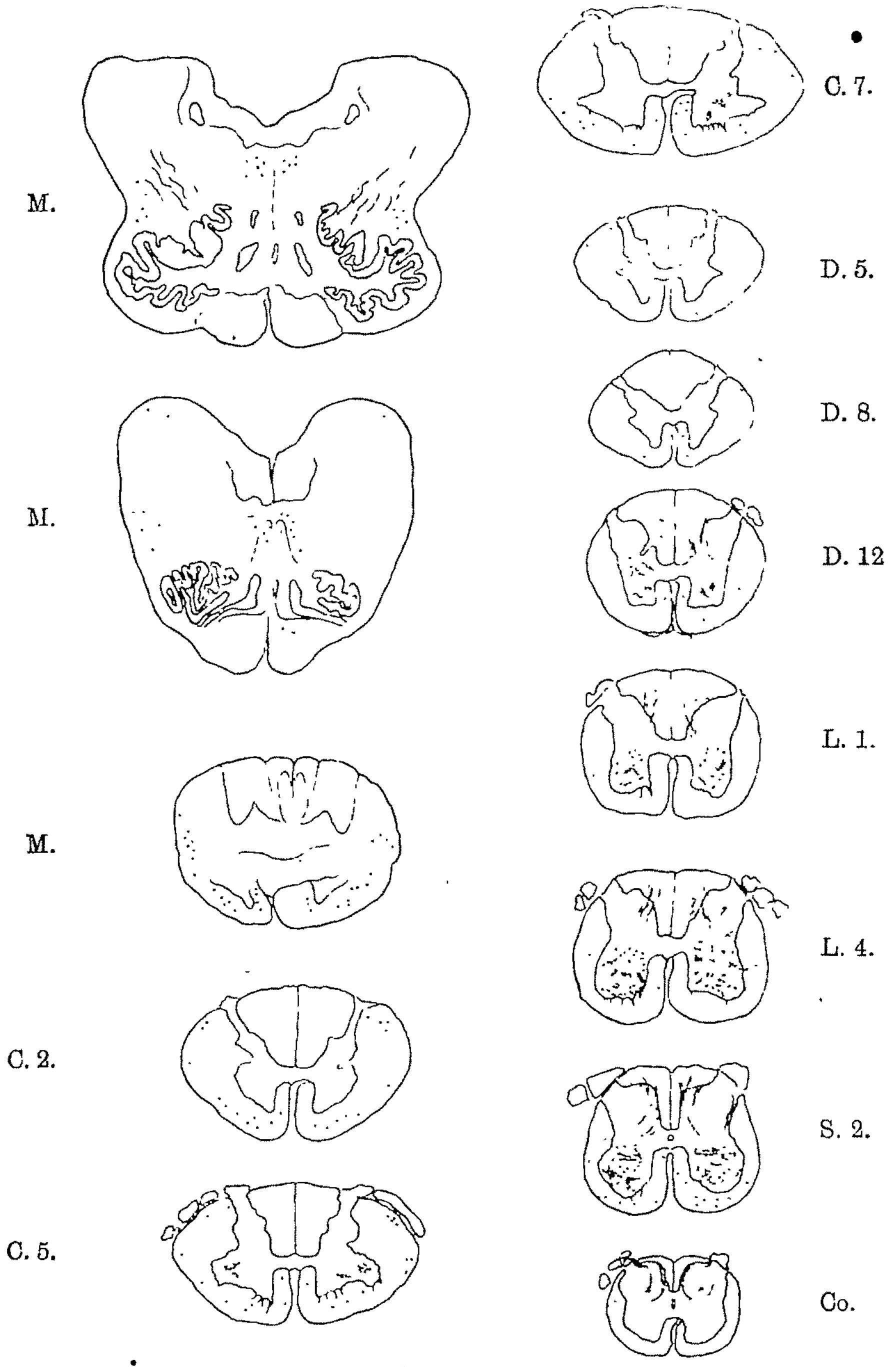
This condition was strictly confined to the anterior horns, and did not involve the white matter of the cord. After section of the cord, this gelatinous material shrank away from the level of the section, so that a depression appeared in the situation of the anterior horns.

Microscopical examination.—Sections of the cord were stained by the Marchi, Weigert-Pal, Nissl and van Giesson methods.

On examining sections stained by the Marchi method, it was found that an area of softening was present in the region of the anterior horn extending through the lumbar region from the twelfth dorsal to the upper sacral segments on both sides. In this area of softening were seen large numbers of fat granules; some of these were especially numerous in the perivascular lymphatics, and marked out the course of a vessel, while others were distributed throughout the area of softening (plate II., figs. 1 and 2).

At the level of the fifth and seventh cervical segments, small areas of softening similar to those in the lumbar region could be seen occupying a situation in the anterior horn (plate III., figs. C. 5 and C. 7).

PLATE III.



Scattered throughout the antero-lateral tracts were numerous degenerate fibres; these could not be said to be confined to any one tract, although in the upper thoracic and cervical region they tended to limit themselves to the direct cerebellar tract and anterior portion of the white matter, leaving the crossed pyramidal tracts less affected (plate III., figs. C. 2, 5, 7, D. 5). This degeneration could be traced up to the direct cerebellar tract, and to the region of the posterior longitudinal bundle in the medulla. A few degenerate fibres could be seen in the posterior columns in the lumbar region, but they did not form any definite tract.

It may here be noted that an essential chemical difference in the constitution of the fat which occurred in the gray matter and that which occurred in the white must exist, for the fat of the gray matter, which was intensely stained by osmic acid, rapidly lost its blackness when mounted in xylol canada balsam, whereas the stained fat in the white matter remained unaltered.

In section stained by the Weigert-Pal method, the most striking naked-eye feature was the difference in staining between the posterior and antero-lateral tracts. The former were darkly and well stained, while the latter were pale. The same points, however, could be observed with regard to the crossed pyramidal tracts, as was noted by the Marchi method, viz., that they were less affected than other parts of the antero-lateral tract (plate IV., fig. 1). The fibres of the direct cerebellar tract on the outer side of the cord were better stained than those nearer the crossed pyramidal tract, it seems therefore probable that the inner group of fibres arose from the lower segments of the cord (plate IV., fig. 1).

With regard to the condition of the gray matter, the most striking feature was the absence of nerve fibres and nerve cells in the anterior horns in the lumbar region of the cord (plate IV., fig. 2).

By the Nissl method the paucity of all cells in the anterior horns of the lumbar region was the most striking feature; in the same region a few cells were, however, present, and appeared normal in structure. In the lower thoracic region the cells of Clarke's column were affected on the one side, while they were apparently normal on the other. The paucity of cells in the thoracic and cervical region was striking, especially when the relatively good movement that the child had in the arms was taken into consideration.

By the van Giesson method the anterior horns of the lumbar region were seen to be occupied by a tissue composed of fine filaments with comparatively few nuclei, resembling very closely the tissue found in a glioma. There were numerous newly-formed vessels in this area, but there were no signs of acute inflammation.

The walls of the anterior median artery appeared rather thicker than normal, but otherwise the vessels appeared natural. The white matter and the posterior horns of the cord appeared normal.

The muscles of the legs showed most extensive atrophy, practically no normal muscle fibres existing, the biceps muscle of the arm contained both atrophied and normal muscle fibres (plate V., fig. 2). The muscle spindles were well preserved, and the intrafusal fibres were normal, thus corresponding to the condition which has been shown to be present in the muscles of cases of infantile paralysis (BRAIN, vol. xx). The nerves within the muscle spindles were normal.

The nerve trunks (sciatic and anterior crural), considering the extreme atrophy of the muscles, contained a large number of normal nerve fibres (plate V., fig. 1); but it will be noticed that the majority of these fibres are of large size, and almost certainly sensory and connected with the posterior roots.

Case 3.—The third case is that of a boy, aged 4 years, who was admitted to the hospital for sick children, under the care of Dr. Barlow, suffering from tuberculosis. He died of tuberculous meningitis.

Two years before admission to the hospital he had an attack of infantile paralysis, losing the use of both legs and the left arm. The left arm and the left leg rapidly recovered, but the weakness of the right leg persisted, and there was marked wasting of the right thigh and leg. The knee-jerk on the right side was obtainable, while that on the left side was brisk.

The spinal cord at the time of the *post mortem* appeared normal when, however, after hardening, the roots of the cauda equina were carefully separated it was seen that some of those on the right side had a gray appearance, and were thinner than the corresponding roots of the left side; this was most marked in the first sacral root.

The cord was stained by the Weigert-Pal and by the Nissl method.

On examining sections stained by the Weigert-Pal method at the level of the third lumbar segment, it was noticeable that the anterior horn on the right side was smaller than on the left, and that the whole of the white matter of the antero-lateral column stained less intensely than the white matter of the posterior columns (plate IV., fig. 3).

Under the microscope the difference between the number of medullated fibres in the gray matter of the anterior horns was the most striking feature, and in one part of the horn practically no medullated fibres existed. The difference in the emerging fibres of the two sides to form the anterior roots was also striking, for on the one side they were well stained, while on the other they were pale and atrophied. With regard to the cells of the anterior horn, the greatest diminution appeared at the level of the third lumbar segment, but even in this region one or two groups of well-formed cells were present. The roots on the affected side were seen to contain far fewer normal fibres than the unaffected side.

DISCUSSION.

The three cases above recorded illustrate the changes which are found in acute anterior poliomyelitis at various stages of the disease (so far as the bacterial cause of the disease is concerned nothing has been learnt).

In the cord of the child who died fourteen days after the onset of the disease, the acutest inflammation was present in the gray matter of the anterior horns, with a large amount of small round cell exudation, hæmorrhages and thrombosis of vessels. In the second case an area of necrotic tissue limited to the anterior horns in the lumbar and sacral regions. In the third case a diminution in the size of the anterior horn of the affected side due to cicatricial contraction.

The question at once arises, what is the cause of this affection of the anterior horn which in the first place gives rise to the acutest inflammation, then to necrosis, and finally to cicatrisation?

Is the condition due to a primary inflammation, followed by a thrombosis of vessels, or is it primarily due to a thrombosis of vessels followed by inflammatory changes and hæmorrhage, for certainly so far as the present series of cases is concerned, the view that this condition is due to a direct affection of the cells of the anterior horns may be dismissed?

Both theories have had their advocates, and quite recently Head and Campbell have strongly expressed the opinion that the thrombosis is secondary to an acute infection probably of bacterial origin; their argument is as follows, and may well be given in their own words:—"If by 'vascular' is meant that the disease is directly secondary to occlusion or interference with a vessel we deny its correctness, if, on the other hand, it is meant that the vessels play an important part in the general inflammatory process it is a *platitude*. Many observers have occluded the vessels of the spinal cord for varying periods, and examined the changes so produced in the cells. Such occlusion of the arteries produces marked chromolytic change in the cells of the spinal cord, but is not followed by inflammatory change or hæmorrhages. Leonard Hill ligatured the cerebral arteries in monkeys, and the brains of these animals were examined by Mott at varying periods after the operation. Here, again, extreme chromatolysis of the cortical cells was found, but no inflammatory change or hæmorrhages."

Such arguments sound almost conclusive, but it must be remembered that obliteration of a large vessel is by no means necessarily comparable to the condition which is produced when thrombosis occurs in smaller vessels. Far more to the point are the experiments of Prevost and Cotard (although performed many years ago) with regard to the changes which take place in an infarcted area. By the means of fine tobacco seeds they succeeded in producing infarcts in various organs, and they proved by their experiments that congestion, hæmorrhages and exudation of cells were the early results of obliteration of small vessels. They enter fully into the mechanical explanation of this congestion, but it is unnecessary here to deal with the somewhat prolonged discussion on that point, but the fact that exactly similar changes such as are found in early cases of infantile paralysis, viz., hæmorrhages and small cell exudation, may be produced by obliteration of small vessels at once negatives Head's and Campbell's assertion that "inflammatory changes" are not the result of thrombosis.

Having thus established the possibility of such a condition as is here present being due to primary occlusion of vessels, it remains to be seen what evidence there is that the condition may be due to the obliteration of any one vessel or set of vessels. The gray matter of the anterior horns receives its chief blood supply from branches of the anterior median artery. Branches from this artery come off at the lowest part of the anterior median fissure, and entering the gray matter of the anterior horn supply the whole of the gray matter on the ventricle side of, and including the column of Clarke, but not the posterior horns nor the white matter of the cord. Now, in the spinal cords from cases of infantile paralysis the line of demarcation between the affected and unaffected portion is sharply defined not only in the acute cases, but also in the cases of longer standing. It cannot be denied, however, that the vessels in the ventral portion of the spinal cord outside the gray matter are engorged, but there is no softening such as is present in the gray matter. The line of demarcation in cases of infarct in which the acute stage has passed is always sharp and well defined, and so is the area of softening in the anterior horn of infantile paralysis, and, when one remembers how closely the area of softening corresponds to the distribution of the branch of the anterior median artery, it is but reasonable to suggest the great probability that such a condition is due to an occlusion of a vessel or vessels.

Again, if the condition which is present in the cord of a child who has suffered from infantile paralysis be compared with that due to the thrombosis of small vessels in the brain, the conditions are strikingly similar.

Further, the occurrence of the disease most frequently in the lumbar region seems to be another point greatly in favour of the view that the condition is primarily vascular, for it is known that the gray matter of the anterior horn of the lumbar region is situated at a point most peripheral from its blood supply, which is derived from the vertebral artery, the blood has therefore to travel the whole length of the spinal arteries, and, further, it has been shown by Moxon that the reinforcing arteries of the roots do not readily assist in the supply of this part of the cord, for they cannot be injected through the femoral artery.

From a clinical standpoint it might be argued that the

PLATE I.

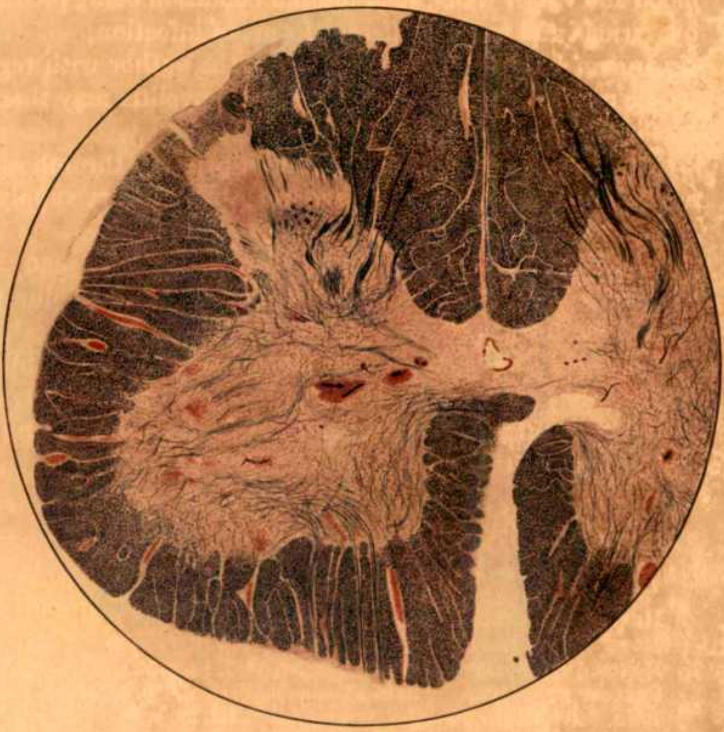


PLATE II.

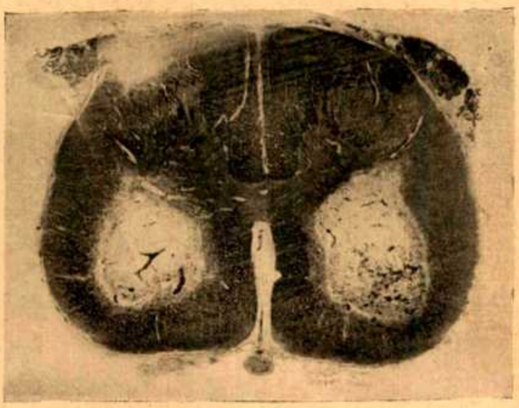


FIG. 1.

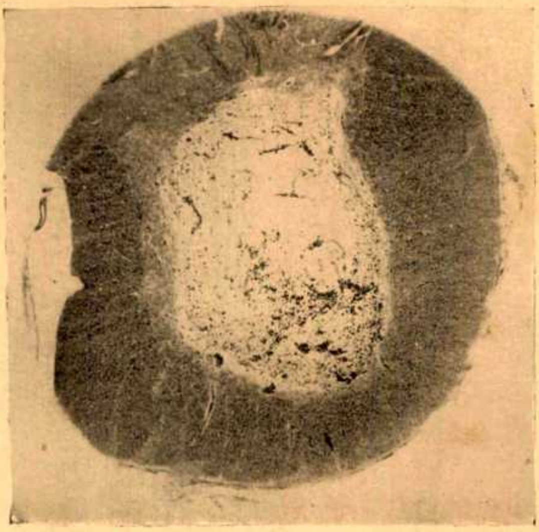


FIG. 2.

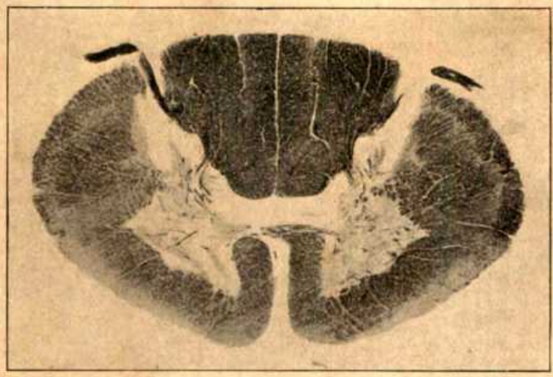


FIG. 1.

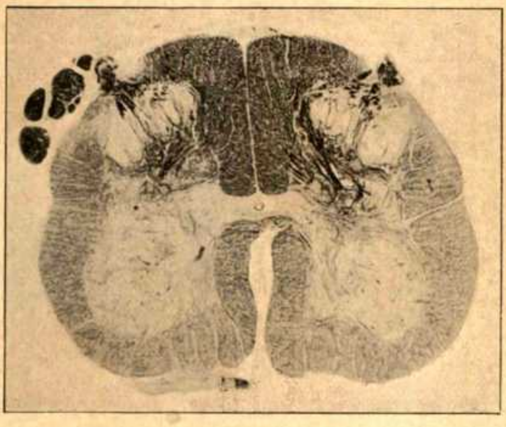


FIG. 2.

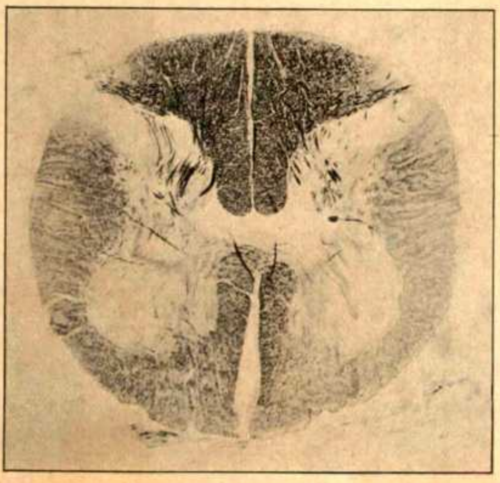


FIG. 3.

PLATE V.

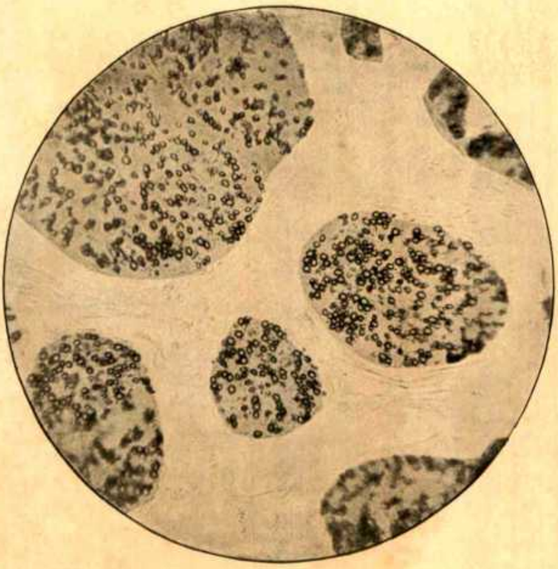


FIG. 1.

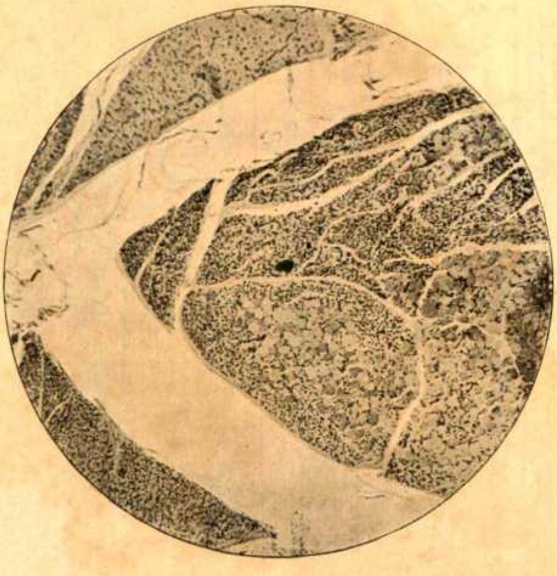


FIG. 2.

ON SO-CALLED FACIAL HEMIHYPERTROPHY.

BY H. MACKAY, M.D., DEVIZES.

THE extreme rarity of this condition is a sufficient reason for recording a fresh case, and for recapitulating what is known of it.

Described first by Beck in 1836 (1) it was nearly thirty years before a second case was reported by Friedreich (2). The Heidelberg professor described a third case observed by Heumann (3), and to these, Passauer in 1866 (4) added a fourth.

In 1869 Trelat and Monod (5) published a monograph on the condition of unilateral hypertrophy, partial or complete. The authors collected twelve cases of this condition, some of which, viz., those of Devouges (6), Ollier (7), and Monod (8), have been referred to since as instances of facial hemihypertrophy. I have not included them in the present series of cases, however, because the original observers state regarding each case that the facial asymmetry was insignificant, or that it eluded measurement.

The next contribution of importance was that of Fischer, on "Giant Growth," in 1879 (9). Fischer reported a fresh case of facial hemihypertrophy under his own care, and endeavoured to classify the various cases of hypertrophy that had already been reported.

He distinguished between "true" and "false" hypertrophy, limiting the former term to those instances in which all the tissues of a part were equally enlarged, and the part remained functionally useful, and assigning the latter to those in which the enlargement was due to, and was restricted to, over development of one tissue only.

The division was unsatisfactory and is now obsolete. It left unclassified the cases in which more than one tissue, *e.g.*, fat, sebaceous glands, and connective tissue have undergone hyperplasia, but in which all the tissues of the part, *e.g.*, the bones, have not participated.

While undoubtedly the conditions of lymphangioma and telangiectasis which Fischer appears to have had in view in framing his distinction, ought to be excluded from the list of hypertrophies, yet the latter term has, by the common consent of pathologists, been applied to those cases where hypertrophy or hyperplasia of one tissue, such as connective tissue or muscle, has taken place without exhibiting the intrinsic characters of tumour formation.[1]

Passing over the contributions of Ziehl (10), Schieck (11), and Finlayson (12), each of whom described a fresh case, Lewin (13) in 1884 published an elaborate paper on what he considered to be the correlated conditions of facial hemiatrophy, facial and bodily hemihypertrophy, and cutaneous pigmentation. He gave a list of published cases in each class, including a case of facial hypertrophy of his own. He included in the latter condition, however, two cases published by Stilling (14), to which as cases of facial hemihypertrophy exception may reasonably be taken. Both were cases of swelling of the soft parts of the cheek, in one instance as large as a child's head, in the other as large as a man's fist, which appeared subsequently to injuries in this region. Both were accompanied by extreme dilatation of the blood-vessels of the part; in one case telangiectatic excrescences the size of raspberries being present, while in the other a semi-erectile condition existed, the swelling becoming plainly larger and more distended when the patient bent over.

Inasmuch as local ectases and vascular dilatations are of frequent occurrence in the records of undoubted cases of facial hypertrophy, and it is difficult to decide from Stilling's account how much of the swelling in each case was due to angiomatous conditions and how much to hyperplasia of tissues other than vascular, I have not felt justified in altogether omitting these cases, but have placed them, as doubtful instances, in a division apart.

---

[1] Whether osseous hypertrophy—hyperostosis, pure and simple—of a part such as the face or skull should be placed in a category apart from the condition of mixed overgrowth of bones and soft parts, which, in the majority of instances characterises facial hemihypertrophy, appears to be at present undecided.

Mr. Hutchinson has described (26) as "Unilateral Hypertrophy of the Skull" a case, to be referred to later, which would probably be classified during life as unilateral facial (and cranial) hypertrophy.

Brissaud also (62) has recently described as "hemicraniosis" two cases in which the condition is apparently identical with that described by Mr. Hutchinson. To these cases also reference will be made later.

It is, however, to the Salpetrière school that we owe the most important recent studies of the subject. Sabrazés and Cabannes (15) in 1898 described a fresh case with a bibliography. Their list of published observations, however, requires revision, as they have included as instances of facial hypertrophy, cases (*e.g.*, *Obs.* xiii. in their series) in which the face was not affected. These authors distinguish between the congenital and the acquired cases, and assign to each a different pathogeny. To this point reference will be made later.

Still later, the teachings of Brissaud on the influence of metamerism in nervous disease, and of Meige on chronic dystrophy, have drawn the attention of many of their pupils to the subject of hypertrophy of limbs, and other portions of the body. Unilateral hypertrophy of the face has also come within the purview of these studies.

The question whether facial hypertrophy, in any form, may be considered as a dystrophy allied in its nature to trophœdema or not is yet an open one. Meige himself (16) adopts towards the suggestion an attitude of caution.

As regards several instances of facial hemihypertrophy, however, *e.g.*, those of Beck, Berger, and Ziehl, in which the facial enlargement has concerned the soft parts only, and has exhibited the characters of a swelling, not pitting on pressure, colourless, and painless, it would appear to be difficult to separate the condition clinically from that described as "chronic trophœdema" of Meige. It is for this reason that I have included in the list of cases given below, the examples described by Rapin (17), Hertoghe (18), and Thomas (19).

English and American observations of the disease have been scanty.

Barwell (20) described a case in 1875, and again in 1881.

Heath (21) figured a case in "Injuries and Diseases of the Jaws." Sir Geo. Humphrey (22) described and figured a doubtful case, in which the gums were chiefly concerned, in 1886. In each of these cases it was the surgical rather than the neurological interest of the case that appealed to the observer.

Dr. Montgomery, of San Francisco (23), in describing a case, published a brief but excellent résumé of the subject in 1893.

Mr. Hutchinson and Dr. Alexis Thompson (26) have each reported cases of what the former describes as "Unilateral Hypertrophy of the Skull," and have admirably described, from skulls in their possession the osteo-pathic changes which occur. As regards the facial condition of these patients during life, the details are not sufficiently full to allow of their being tabulated with the other cases here presented. While it is in doubt, however, whether and to what extent the soft parts were involved, I think there need be no hesitation in including them as cases of facial (and cranial) hemihypertrophy.

Brissaud and Lereboullet (62), in the cases already alluded to above, describe a condition of hyperostosis of one half of the face and skull in the region of the trigeminal nerve, for which they propose the distinctive title of "hemicraniosis."

In the first case epileptic attacks commenced at the age of 20 in a patient in whom, from the age of two months, there had been noticed asymmetry of the face, with bossing of the left frontal bone. Headache, but no vomiting, was present, and the fundus was normal.

In the second, the enlargement of the face and skull had also existed from childhood. The right half of the face was hypertrophied, and there was a well-marked painless boss in the right frontal region. Latterly, there ensued vomiting, headache, strabismus, double choked disc, exophthalmos, and ultimately complete blindness, leading up to exitus lethalis.

The autopsy showed the presence of numerous small angiolithic tumours on the inner surface of the dura on the right side, and multiple exostoses, the size of nuts, on the inner aspect of the cranium on the same side, which corresponded to correlated depressions in the cerebral substance.

The authors claim for these cases a certain relationship to facial hemiatrophy, inasmuch as in each condition there is present a unilateral trophic affection in the region of the trigeminal.

The condition thus depicted appears to be identical with that described by Hutchinson and Alexis Thompson as unilateral hypertrophy of the skull, and by other observers as facial hemihypertrophy, but, in deference to the distinctive nomenclature adopted for it by so distinguished an authority as Brissaud, I have excluded these observations from the subjoined list of cases of facial hypertrophy.

In the cases here given, the original records have been consulted in all instances, with the exception of Cases 1, 14, and 22.

In addition to the observations of Ollier, and Monod, already referred to, I have omitted several others included by former writers, on account of the poverty of detail in the available records. Such are those of Steffen (27), Zhukovsky (28), and Stembo (29), quoted by Messrs. Sabrazès and Cabannes, and that of Logan given in the *Virchow-Hirsch Jahresbericht* for 1868.

Of the associated, but commoner, condition of unilateral hypertrophy of the body, the literature is too voluminous to attempt to refer to it here.

Case 1.—BECK. *Medicinische Annalen von Puchelt, Chelius und Naegele*, 1836, Bd. II., No. 1, p. 89 (quoted by Ziehl—*Virchow's Archives*, Bd. xci.).

A girl of 3 years presented a hypertrophic fulness of the right cheek, which had been present since birth, and increased with the growth of the child. The soft parts alone were affected.

Case 2.—FRIEDREICH, 1863 (quoted by Ziehl), *Virchow's Archiv.*, Bd. xxviii., p. 474.

A girl of 16 showed soon after birth enlargement of the right side of the face, chiefly marked in the infraorbital portion. The forehead and eyes were symmetrical. The external ear was enlarged in all directions. The hypertrophied side showed circumscribed patches of pigmentation, and an enormous development of abnormal hairs existed on the ear, cheek, temple, and along the jaws. The hairy scalp and the hands showed no abnormal development in the hairs. The irides showed similar coloration. An exaggerated sebaceous secretion covered the neighbourhood of the ear and mastoid with a greasy layer.

On the right side the alveolar processes of the upper and lower jaw were double the thickness of the opposite side; the teeth also were larger, longer, and thicker than those on the left. Toothache was frequent.

The right tonsil and right half of the tongue were much enlarged, and the saliva was more abundant on this side. On the hypertrophied half of the tongue sensibility to taste and touch were less developed. The right cheek was of a deeper colour than the left, and experienced a subjective sensation of warmth. The child suffered from otorrhœa on the right side.

An autopsy was obtained, but nothing throwing any light on the condition was found. The nerve trunks at the base of the brain were unaltered (macroscopically). The petrous portion on the right side was hypertrophied, and showed exostoses projecting from its surface which had indented and compressed the opposing surface of the brain.

Friedreich also communicated the following case:—

Case 3.—HEUMANN (*loc. cit.*).

A girl, five years old, had shown since birth enlargement of the left side of the face, which involved the cheek, the two jaws, the teeth and the tongue. The skin of the ear was thickened, and the hairs more developed. There had been a fœtid otorrhœa from the left ear since birth, but the hearing was not impaired. There was a constant flow of saliva from the left side of the mouth.

The child died from meningitis following caries of the petrous bone. No autopsy.

Case 4.—PASSAUER, 1866, *Virch. Archiv.*, xxxvii., p. 410.

A boy of 11 showed congenital hyperplasia of the left side of the face. During pregnancy the mother was crushed by a wagon against a wall, and suffered severe abdominal pain for some time.

The enlargement involved the cheek, upper and lower lips, and the chin, and was limited by the median line of the face. The alveolar borders of the upper and lower jaws were much enlarged on the left side, as also was the left half of the tongue and palate, and the teeth. On the hypertrophied portion of the face there was no play of expression.

Case 5.—BERGER. "Zur Aetiologie und Pathologie der sogenannten Muskelhypertrophie." *Deutsche Archiv. für Klin. Medicin*, 1872, p. 393.

In a woman of 58, Berger noticed hypertrophy of the face supervene in the course of severe and obstinate trigeminal neuralgia. The hypertrophy concerned the soft parts only, and especially the subcutaneous connective tissue.

Case 6.—FISCHER. "Der Riesenwuchs." *Deutsche Zeitschrift für Chirurgie*, Bd. xii., 1879.

In a boy of 7 months the left side of the face had been hypertrophied since birth. The enlargement and thickening affected all the tissues. The skin showed normal structure, and the bones were smooth. Of the latter those chiefly affected were the upper jaw and the frontal bone. The circumference of the head in the frontal plane was 56 cm., of which the left half measured 31, and the right 25 cm. The tongue was normal, and the hairs of the scalp, eyebrows, and eyelashes were equally developed on both sides. There was no difference in the eyes, or in the lachrymal secretion of the two sides. No discolouration was present. Motility, sensibility and temperature, were normal.

Case 7.—BARWELL. *Transactions of Clinical Society*, vol. xxxii., 1881.

A boy of 13 showed enormous hypertrophy of the right side of the face. It affected the head and face. (It appears to be chiefly infraorbital in the illustration. H.M.)

The texture was soft and yielding.

Near the ear was a patch of dark horny ichthyosis (true hypertrophy of the cuticle), which was increasing in size.

The upper and lower maxillæ were massive and thick, and the teeth were hypertrophied also. The tongue was enlarged on the affected side.

Mr. Barwell suggests a perverted action of the sympathetic as a probable cause. The common carotid was tied, and the patient died from secondary hæmorrhage.

As an appendix to the above may be quoted a previous observation of Mr. Barwell's recorded in the *Medical Times and*

Gazette, vol. i., 1875, which, though not unilateral, possesses considerable interest. The patient was a lad of 19, who showed hypertrophy of both sides of the face. It affected the soft parts only, the bones being of natural size and shape. It was stated to have appeared in a single night.

Mr. Barwell tied both facial arteries, but without much improvement.

Case 8.—Ziehl. *Virchow Archiv.*, Bd. xci., s. 92, 1883.

A boy of 4 years presented hypertrophy of the right side of the face which had existed since birth. Four or five weeks before her confinement the mother had been stung by nettles on the right side of the face. At five weeks old the child was convulsed, due to hydrocephalus. Subsequently fits were frequent. The enlargement affected the lower half of the right side of the face. The brow, skull, eyebrows, eyelids, and lashes were unaffected, but the hair on the right side was stiffer and coarser than on the left. There was convergent squint of the right eye with prophthalmos. The right iris was larger than the left, and the right ear longer and broader. Brown pigment spots were present at many places in the face. The alveolar processes of the upper and lower jaw were enlarged, but the teeth were equal on both sides. The right half of the tongue and right tonsil were much enlarged, and there was increased salivary flow from the right side of the mouth.

Motility and sensibility normal. The right hand was larger than the left.

Case 9.—Schieck. "Ein Fall von einseitiger Gesichts-hypertrophie," *Berlin Klin. Wochenschrift*, 1883, p. 200.

The patient was a girl 9 years old, born of a family with no neuropathic tendency. Nothing abnormal was noticed at birth.

At the end of the second year there appeared an increasing swelling of the left cheek and upper lip. At the same time further development of teeth in the upper jaw of the same side ceased. The child was obtuse mentally. At 9 years the overgrowth affected the soft parts of the left cheek and upper lip, and to a less degree the left lower lid. The left nostril was thicker than the right. The lower part of the face and the philtrum of the upper lip were pushed over to the right side.

The left cheek was redder, and the growth of hair distinctly stronger. Sweat and sebaceous secretion not increased. Of the bones the zygoma, the body of the left maxilla, and particularly the left upper alveolar process were more strongly developed than the right. The left upper jaw contained only the two incisors, canine and first premolar. The lower jaw presented nothing abnormal.

The left half of tongue and left tonsil not enlarged. Left ear and brow normal. Growth of hair on both sides of skull equal, as also pupils. Motility and sensibility normal.

Case 10.—EVE. *Transactions Pathological Society*, vol. xxxiv., 1883.

A woman of 34 had congenital hypertrophy of the foot and leg on the left side, which had rapidly increased.

The left half of the head and face were also larger, as was the left half of the tongue.

The autopsy showed symmetrical enlargement of the left side of the brain, with several osseous growths, the size of peas, in the dura. A fibrous tumour, the size of a filbert, grew from the outer side of the left cornea, and an epulis, the size of a pigeon's egg, from the left upper jaw. The left patella showed hyperostosis.

Case 11.—FINLAYSON. "Case of a child affected with congenital unilateral hypertrophy. — *Glasgow Medical Journal*, November, 1884.

A female child of 18 months had suffered from repeated attacks of otorrhœa in the right ear. From birth the right side had been hypertrophied throughout. The facial enlargement affected the bones as well as the soft parts. The parietal protuberance was excessively developed, as also were the upper and lower jaws. The tongue on right side and right tonsil were larger. The palate and uvula were normal. The temperature on the right side of the face was 3° to 4° higher than on the left. Teething was precocious on the right side.

On the left side the index, middle, and ring fingers were hypertrophied. Most excessive, however, in size was the middle finger of the right hand.

Under the excitement of crying, patches of colour, red, purple, or blue, come out over the skin in the vicinity of the navel, on the inner and posterior aspects of the hypertrophied right leg, and on the back. The patches suggest cutaneous nævi. The discolouration fades as the child becomes quiet.

Case 12.—Lewin. "Studien über die bei halbseitigen Atrophien und Hypertrophien, namentlich der Gesichts vorkommenden Erscheinungen, &c."—*Charité Annalen*, Bd. ix. s 619, 1884.

A man of 26 showed crossed hypertrophy, affecting, on the left side, the face, neck, and arm, and on the right side the leg, which was longer and thicker than the left.

In the face the hypertrophy concerns the soft parts, and to a less extent the bones, viz., the frontal, zygoma, and upper and lower jaws. The hypertrophy is limited, vertically by the middle line, and otherwise by a line from the middle of the brow to the upper insertion of the auricle.

The skin over this area is hyperæmic, of a bright red colour, and hyperidrotic. The conjunctival and pharyngeal mucous membrane are also hyperæmic, and so is the left half of the hard and soft palate. Tongue and teeth normal. Hyperæmic patches are present on the chest, and flecks of vitiligo are present on the left half of the abdomen, reaching to the middle line, but not passing beyond it. Nævi melanoides are present on the left side of the neck. The sensibility to temperature is diminished on the right side, and general sensibility on the right side of the tongue only.

Left-sided toothache had been frequent since childhood.

The temperature was lower by $\frac{6}{10}$° C. in the left axilla, though the patient had often a subjective sense of warmth on this side.

Case 13.—Humphrey. "Annals of Surgery," vol. iii., 1886.

A boy of 12 suffered from unilateral hypertrophy of the gums of the lower jaw on the left side, which were thicker than those on the right.

There was no discoverable difference between the bones of the two sides of the head and face, but on the left side the tonsil and soft palate were larger than on the right. The upper and lower lip on the left side, protruded and everted, were thick and coarse. The left eyelid was larger and the eyelashes longer than the right, and the hairs of the left eyebrow were coarser and more numerous. The pinna of the left ear was larger and thicker than the right. The hair of the scalp came down lower in front of the ear, and the skin of the face was fuller and more covered with hair. The papillæ on the left side of the tongue were also larger.

The second digit of the left foot and the right thumb were abnormal, ending in soft bulbous extremities.

Case 14.—MASMÉJEAN. "Thèse de Montpellier," 1887-8 (quoted by Sabrazés and Cabannes).

An infant of 13 months had hypertrophy of the right side of the face, and of the limbs on the right side. The hypertrophy did not affect the length of the limbs, but only their thickness. The augmentation appeared to be chiefly due to an increase of fatty tissue.

Case 15.—KIWULL. "Ein Beitrag fur Casinstik der halbseitigen Gesichts-hypertrophie." *Fortschritte der Medicin*, 1890, Bd. viii.

A girl of 18 showed right-sided hypertrophy of the face, which was congenital, and progressive. It affected both the bones and the soft parts.

There was bossing of the right frontal bone and prominence of the supraorbital ridge. The upper and lower jaws were enlarged and the surface of the latter was irregular and rough. The right alveolar processes were twice as large as the left. The teeth were widely spaced apart, the lips greatly thickened, and the ear enlarged. A zone of hyperæmia extended obliquely to the right from the middle of the nose for a distance of 2 cm. The right half of the tongue was three times its normal size, and the right tonsil also enlarged. From the right angle of the mouth ran a constant flow of saliva. Taste was blunted on the right side of the tongue, but sensibility of the face was unaltered. Portions of the skin and lip were removed for cosmetic reasons.

On examination, the various layers of the epidermis and cutis were of normal thickness. The sebaceous glands were hypertrophied, and the hair follicles normal. The fatty and connective tissues were increased in bulk, the former beneath the skin, and the latter beneath the mucosa. This latter, made up of fine fibres with few cells, was traversed by dilated veins and arteries. The muscle fibres took but slight part in the overgrowth.

Case 16.—TILANUS. *Munchener Medicinische Wochenschrift*, No. 4, 1893.

A girl of 10 had hypertrophy of the right side of the body, including the face. It was not noticed at birth, nor till the child was 3 years old. It had increased considerably of late.

The facial enlargement concerned the cheek, which was thicker and wider, and the right half of the tongue. The cranial circumference was also increased on the right side. The right shoulder, arm, half of trunk, buttock, leg and foot, all showed overgrowth. The musculature on this side was more powerful.

Electrical reactions and reflexes normal. There was a reticulated marbling of the skin of the trunk and limbs. Under the influence of cold the colour of the marbling became darker, and even blue.

The author thinks it possible that a central cause may be at the root of the overgrowth, and refers to the case of Demme.

Demme's case (*Wiener Medicinische Wochenschrift*, No. 2, Jahrgang xiv., 1891) was one of congenital unilateral hypertrophy in a baby girl who lived only a few days. The hypertrophy affected the whole of the left side of the body. On autopsy the cross sections of the cerebral hemisphere on the left side were larger than those on the right. The muscle fibres on the left side were hypertrophied on cross section, and the nuclei of the sarcolemma increased in number, and arranged in rows.

Case 17.—MONTGOMERY. *Medical News*, vol. ii., 1893, p. 61.

The patient was a man of 31, of German extraction. At 2 years he had brain fever. At 8 or 9 an alveolar abscess formed on the left lower jaw and caused much pain. No neuralgia. No abnormality of dentition.

A photograph taken at 5 shows the face to be perfectly symmetrical.

At 10 the gums were noticed to be enlarged, and at 12 the deformity was noticeable on the left side of the face.

At 31 the deformity occupied an irregular quadrilateral enclosed internally, by the mesial line from the anterior extremity of the sagittal suture, down to the mental symphysis; below by the border of the lower jaw and on to the tip of the mastoid, and posteriorly from the mastoid to the sagittal suture. The hypertrophy is most marked in the supraorbital, molar, and mental regions, where the tissues are raised up into tumefied masses consisting of soft parts and bone. The frontal bone shares in the hypertrophy, a mass of bone running upwards and backwards over this region from the outer extremity of the supraorbital ridge.

The scalp and hair are normal. The upper half of the left ear is hypertrophied. The discs are excavated, and not engorged. The left lower jaw is enlarged and roughened. No enlargement of teeth, tongue, or tonsil. No sensory abnormality, general or special. The deformity is progressive.

Portions of skin removed for æsthetic purposes showed, on section, increase in the size of the hairs and sebaceous glands. Also plugging of the sebaceous glands with sebum, and degenerated cells. There was no increase in connective tissue, or fat.

Case 18.—DANA. *Journal of Nervous and Mental Disease*, November, 1893, page 234.

The patient was a professional giant of 19. Height 7 ft. 4 in., weight 325 lbs.

Acromegaly was present, the hands and feet being enormous. He " wore a shoe just a foot and a half long." The enlargement was symmetrical in both upper and lower extremities. The left half of the face was hypertrophied, the enlargement having begun at puberty, and being progressive. It corresponded closely with the distribution of the left trigeminus, and involved the upper and lower jaw, the alveolar processes being enormously enlarged, as also the palatal arch. The teeth were normal, and so were the orbits, but the left frontal bone showed great bulging.

No pigmentation. Muscular system moderately developed, but the patient was weak. Vision good. No contraction of fields. Pupils normal. Intelligence good. Appetite prodigious.

Case 19.—HEATH. " Injuries and Diseases of Jaws," 1894, page 136.

A boy of 12 showed enlargement of the face, which was noticed at three months old. It affected the bones and soft parts on the left side.

The superior maxilla on this side retained in its alveoli the temporary incisors and canines, but these had been replaced by permanent teeth on the sound side.

Mr. Heath removed the superior maxilla, but the patient's condition, two years later, was as unsightly as before.

Case 20.—ARNHEIM. " Hypertrophie congénitale de la moitié droite de la face et du bras correspondant."—*Semaine Medicale*, 1895 and 1897.

A girl of 4 months exhibited congenital hypertrophy of the right side of the face and the corresponding arm. The hyperplasia of the face formed a prominent mass, which raised the left lip.

The right half of the tongue was enormously hypertrophied, and the right ear was larger by 1 cm. than the left. The right arm also longer than the left.

Dyspnœa was present, of cardiac origin.

At 2 years the child died of broncho-pneumonia. The autopsy showed that the hypertrophy extended to the internal organs on the right side of the chest. The right auricle and ventricle were larger than the left, and the right bronchi more spacious.

Case 21.—Sabrazès and Cabannes. "Hemi-hypertrophie faciale."—*Nouvelle Iconographie de la Salpêtrière*, tome xi., 1898.

A boy of 3 years presented congenital hypertrophy of the right side of the face.

At the fourth month of pregnancy the mother suffered a fall on the abdomen, and at the seventh an emotional shock. At four months the child had a purulent otorrhœa (right), which lasted two months, and left impaired hearing. Dentition was precocious on this (right) side. The original disproportion has been maintained since birth.

It concerns the bones and soft parts, and the frontal boss is enlarged. The cranium is asymmetrical, showing increased development and fulness on the right side. Venous dilatations are present on the mucous surfaces of the left cheek.

In the median line in the occipital region there is present in the scalp a pigmented streak, cafe-au-lait colour, extending from the nape of the neck to four fingers' breadth below the summit of the vertex; over this streak the hair exhibits a negroid curliness.

Electrical examination shows on the left a normal reaction, and on the right a diminution of excitability, owing to the increased resistance of the tissues.

Sensibility to pain is diminished slightly on the affected cheek. On the forehead it is the same as on the opposite side. The gustatory sense is normal. The pharyngeal and palpebral reflexes are normal.

The right cheek always redder than the left, becomes scarlet when the patient approaches the fire. In winter it is more sensitive to cold, and suffers from cracks.

The right hand is slightly larger than the left.

Case 22.—Hermanides. "Een geval van hemi-hypertrophia congenita."—*Psychiatr. en neurol.*, *Bladen*, 1899. (Abstract in *Mendel's Centralblatt*, 1900, p. 912.)

In a child of 13 months all parts of the left side of the body were larger than the right. The palpebral opening, eyeball, and pupil, appeared to be somewhat larger on the left side. As a rule the left ear was redder than the right. A striking difference obtained between the two halves of the tongue, but the small right half showed fibrillary twitchings, and the mucous membrane was thrown into small folds. Trophic signs were absent, apart from the increased reddening of the left ear. The teeth and hair on both sides were equally developed. Sensib[illegible] equal on both sides, and no difference could be detec[illegible]sibility.

It appeared, at first, doubtful whether in this case the condition was one of left hypertrophy or of right hemiatrophy, but the author is satisfied that the former was the real condition.

Case 23.—HUTCHINSON. *Polyclinic*, July, 1901.

The patient was a middle-aged woman with unilateral hypertrophy of the face and skull. The soft parts did not appear excessively increased, but there was enlargement of most of the bones in the right half of the face. The frontal boss on the right side was specially prominent, and the right eye bulged forward, probably on account of a growth behind it.

There is no statement as to whether the condition was congenital or not.

Elsewhere the patient showed multiple cutaneous and subcutaneous fibromata scattered over the chest, and also osseous enlargement of the left side of the sternum, involving possibly some of the ribs.

Case 24.—RAPIN. "Sur une forme d'hypertrophie des membres (Dystrophie conjonctive myélopathique)." *Nouvelle Iconographie de la Salpêtrière*, No. 6, 1901.

The case was a congenital one, in a woman of 29. The hypertrophy concerned the right side of the face and the two upper limbs. In the face it affected the cheek and ear on the right side. The maxilla showed no thickening. The teeth were bad. There was no nævus or vascular tâches.

The two arms were equally enlarged. The hypertrophy commenced about the middle of the limbs, but was most marked in the hands. The skin was white, soft to the touch and normal. There were no abnormal vessels on the surface. Under the influence of cold it became quickly cyanotic and stiff.

Palpation gave the impression of moderately firm fatty tissue. It did not pit on pressure. Cutaneous sensibility was normal.

Case 25.—HERTOGHE. "Contribution a l'étude de trophœdème chronique." *Ibid.*, page 497, 1901.

A girl of 21 shows congenital hypertrophy of the right cheek. The tongue is not increased in size, but the papillæ on the affected side are hypertrophied. The teeth are more strongly developed on the affected side. On this side the wisdom tooth is through and well established, but not on the healthy side. The palate o[illegible]ht side is larger, and its rugosities are more marked. [illegible]n this side are hypertrophied.

The bones of the face are not affected, but the middle turbinate is hypertrophied as compared with the sound side.

A sister of the patient shows infantilism at 15 years, and has the lower limbs "en colonnes," the legs being as thick as the thighs. The mother suffered from lipoma of the arm.

Case 26.—THOMAS. "Deux cas d'hemihypertrophie congenitale du corps." *Ibid.*, page 508, 1901.

A female child of four months has congenital hypertrophy of the face and one half of the body. The hypertrophy affects the right cheek, which is plumper and thicker than the left. The skin also appears thicker. The palpebral aperture is wider on the right, but there is no anomaly in the eyes, ears, nose, or mouth. The right half of the tongue is much larger and thicker than the left. Palate and soft palate normal.

The bodily hypertrophy affects the trunk and limbs of the left side. The bones are enlarged as well as the soft parts, and the muscles also participate. The skin is thicker on the affected side.

The hypertrophy is more pronounced in the thigh than in the leg and foot. Coloration, temperature, and sensibility normal.

Two years later the child was examined again. The hypertrophy was still present, but was much less apparent. Teething was delayed, the child at this date having only six.

Case 27.—THOMPSON. *Transactions Med. Chirurg. Soc. of Edinburgh*, vol. xxi., page 204, 1901-02.

In a young woman of 27 enlargement of the left cheek, with hyperostosis of the left upper jaw and malar bone had existed, probably since birth.

The hyperostosis affected the entire upper jaw, except its alveolar margin, and the downward projection of the alveolar margin caused obliquity of the oral fissure. The nose deviated to the opposite side owing to overgrowth of the nasal process.

Case 28.—FOWLER. *Ibid.*, 1901-2.

A girl of 3 showed hemihypertrophy of the right side, affecting the face, arm, thorax, and leg. The distal parts were more hypertrophied than the proximal.

The temperature was higher on the affected side, and the eruption of teeth was earlier. Numerous capillary nævi were scattered over the body.

Case 29.—Mackay. Unpublished.

A girl of 10 suffers from acquired hypertrophy of the left side of face and skull. The child was normal at birth, and a portrait taken at 5 shows no asymmetry of the face. Measles occurred at 8, followed by earache and otorrhœa, and the latter complaints have been frequent since. There has been no general headache, and no trigeminal neuralgia. Memory and intelligence are good. Bodily symmetry is elsewhere perfect, except that the left hand is somewhat larger than the right. The deformity was noticed first at 6 years of age as a bony enlargement on the head near the vertex. Then appeared a similar swelling in the occipital region. Then came a fulness in the forehead, and, lastly, the cheek began to enlarge.

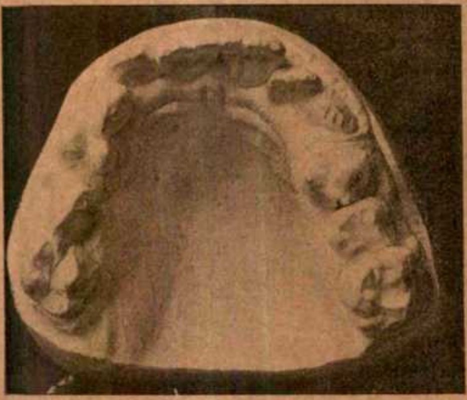


Fig. 4.

Cast of upper dental arch, showing hypertrophy of left alveolar border and teeth. The hypertrophy is sharply defined by the mesial line.

The child is of average build and well nourished. Height 4 ft. 5 in., weight 68 lbs. The bony skeleton elsewhere than in the affected parts is normal. There is no sign of rickets or of congenital syphilis. The teeth are mostly carious. The facial enlargement is limited strictly by the middle line. The left cheek is fuller and thicker, also the left upper and lower lips. Left nasal bone is thickened. Soft parts of nose on left side hypertrophied, and also the left half of the chin.

The bony overgrowth extends to all the left facial bones, and

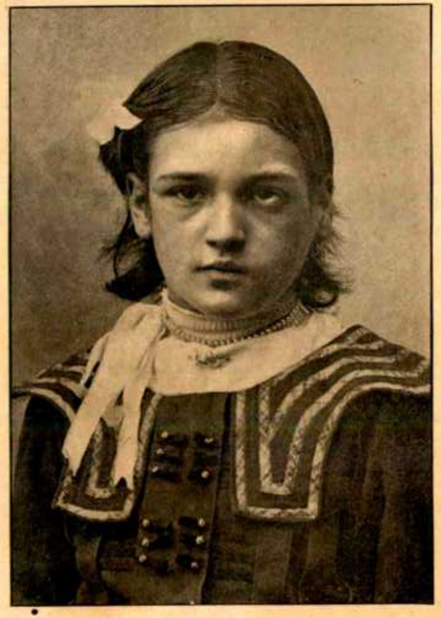


FIG. 1.

Hemihypertrophy of face and cranium.

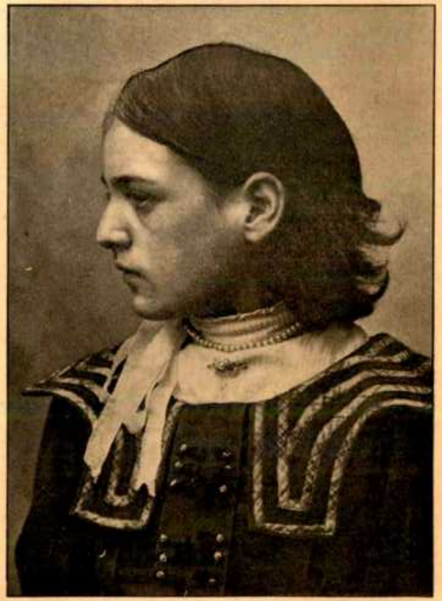


FIG. 2.

Left side view, showing frontal and parietal bossing.

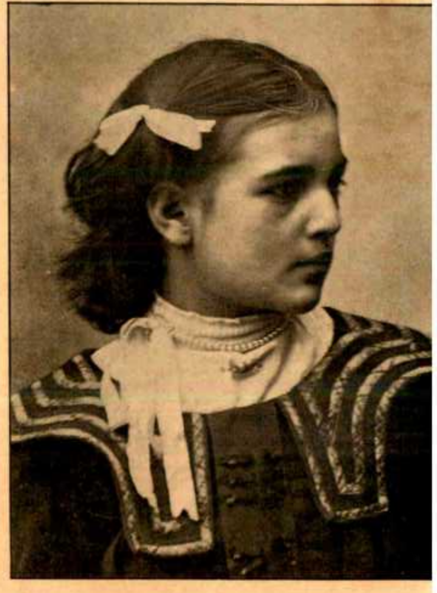


FIG. 3.

Normal side.

to most of the cranial, *e.g.*, the frontal, parietal, sphenoidal (orbital plate, probably), temporal and occipital.

There is marked bossing of the frontal, a smooth prominence of bone commencing at the inner end of the supraorbital ridge, and passing upwards over the middle of the bone to the hairy scalp, where it ends.

Another bony ridge commences to the left of the sagittal suture near the vertex, and runs downwards and forwards over the left parietal bone.

A third mass in the form of a rounded boss occupies the superior angle of the occipital bone posterior to the fontanelle. This boss is mesial in position. In the parotid region the enlargement of the malar and zygoma give rise to a fulness suggesting mumps. The superior maxilla is greatly enlarged, and also its alveolar border. The molars are longer and thicker on this side. Behind the ear the mastoid and the adjoining portion of the temporal bone are enlarged.

Exostoses project from the tip of the mastoid, and from the posterior free border of the superior maxilla.

The posterior part of the skull on the left side is also enlarged, and the hair covering this portion is coarser than on the right. Tongue, palate and tonsils are normal. In the left eye the sclerotic in the lower and outer quadrant of the ball is bulgėd forward, probably owing to some pressure from behind. The rim of the orbit is thickened. Owing to the pushing forward of the outer part of the ball an internal squint, not congenital, has been developed.

Ophthalmoscopic (left eye).—The media are clear, but there is œdematous swelling of the disc, with haziness of the margin, everywhere except in the lower nasal quadrant. The retinal vessels are obscured by the swelling. On the temporal side the retina is swollen and hazy.

Vision R. $\frac{6}{6}$, J.1 at 22cm.; no H.M.
L. $\frac{6}{36}$, J.20; not improved by glasses.

The F. of vision, normal in the right eye, is contracted in the left to the nasal side.

Hearing on the right side is normal, but on the left the watch-tick is heard on contact only. There is an old perforation of the drum.

The special senses are otherwise normal.

Ordinary sensibility to touch, pain, and temperature is unaffected.

Motility and reflexes are normal.

There are no vaso-motor signs, increased sweating, dilated vessels, flushing, or hypersensitiveness to cold. No salivation. There is, however, hypertrichosis of the left cheek in the neighhood of the ear.

Electrical reactions normal.

Stilling's cases are briefly as follows: (" Untersuchungen über die spinal Irritation." Leipzig, 1840, S. 337).

(1) A man of 48 had been thrown when 23 years of age from his horse, injuring his face. Since then the cheek (right) had been larger than the other. Subsequently the same cheek was gored by the horn of an ox. At the time of examination the right cheek was the size of a small child's head. It was soft and smooth and not painful on palpation. At several places small telangiectases the size of raspberries were present. The skin was anæsthetic over an area which was sharply defined. Movement was also lost on the affected side. The tongue and oral mucous membrane were normal. So were the special senses. No pulsation, but the arteries collectively on the affected side, and apparently the carotid, too, were markedly enlarged.

(2) A woman of 38, when nine years old, was struck by a stone on the right cheek, and lost much blood from the mouth. A swelling gradually developed which resisted all treatment. It was the size of a man's fist and limited itself exactly to the right cheek. The skin felt soft, and had the same healthy red colour as the other side. The swollen mucous membrane protruded on the right angle of the mouth showing several enlarged dark coloured blood vessels. Much saliva escaped, which Stilling considered to be serous exudation from the enlarged blood vessels in the mucous membrane. The oral mucous membrane covering the cheek was of a dark blue-red colour, tumid and swollen, with numerous distended blood vessels.

Sensation, motility and reflexes normal. The facial bones were not affected.

When the patient held herself bent over, the swelling became larger and more distended.

The temperature was the same on both sides, and no pulsation was present.

DIGEST OF CLINICAL FEATURES.

Females are affected oftener than males, in the proportion of 17 to 12. The right side is hypertrophied oftener than the left, in the proportion of 15 to 12. This accords with St. Hilaire's (63) opinion regarding unilateral hypertrophy of the body. There is no evidence of hereditary transmission. "Maternal impressions" figure on three occasions.

In twenty-three cases the condition was congenital, or was probably so. In six there is good evidence that it was post-natal, or "acquired." The latter form has appeared at ages varying from 6 to 55 years.

As regards personal antecedents, in four cases (Nos. 2, 3, 21, 29), there has been otorrhœa on the same side as the hypertrophy. In the former three, congenital cases, the otorrhœa could not well have preceded the overgrowth. In the latter case it did. In four other cases there has been present some peripheral irritation of the trigeminal nerve, from frequent toothache (Nos. 2, 12), or from obstinate neuralgia (No. 5), or alveolar abscess (No. 17). In the two former cases the temporal relation of the pain to the hypertrophy is not stated. In the two latter the pain preceded the overgrowth.

In the majority of cases, 19 out of 29, the hypertrophy has been limited to the face, or the face and head. In the remaining ten other parts of the body have been affected also. Of the nineteen former cases, in three (Nos. 1, 5, 25) the soft parts of the cheek were alone affected, in sixteen the facial bones shared in the hypertrophy. In eleven of these the cranial bones were also involved. In three additional cases (Nos. 20, 24, 26) the soft parts of the cheek were hypertrophied, but in conjunction with segmental overgrowth of some other part of the body.

In three cases (Dana's case, and the skulls belonging to Mr. Hutchinson and Dr. Thompson),[1] and also in the two cases of hemicraniosis recorded by Brissaud and Lereboullet, it is stated that the parts affected corresponded exactly to the distribution area of the fifth nerve. As a matter of fact, however, this correspondence with the trigeminal area, either in whole, or in part, appears in every recorded case of facial hypertrophy. In one case only have the limits of the trigeminal been transgressed, viz., in case 29, where an occipital boss was present.

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[1] *Vide infra.*

This relation of hypertrophy to peripheral nerve area is scarcely to be wondered at when we recall that, with the exception of the parotid region, supplied by a branch of the great auricular, the trigeminal supplies practically the anterior two thirds of the cephalon. In the light of Brissaud's (46) observations on the influence of metamerism in nervous disease it is interesting to note that according to Gegenbauer (40), the trigeminal though a single peripheral nerve represents the complete sensory fibres appertaining to the first complete cranial metamere.

As regards the bones, those most frequently hypertrophied were the maxillæ, one or both being enlarged, either in part, or wholly, in 17 cases out of 29. When, as happened in three cases, one maxilla only was affected, it was the upper one that was selected.

In connection with this preference for the maxillary bones it is noteworthy that, as Professor Dixon has pointed out,[1] a similar selection takes place in acromegaly. Professor Dixon calls attention to the fact that the pituitary body is developed partly from the neural tube, and partly from the buccal cavity, and that when this body is diseased, as in acromegaly, the first bones to be enlarged or affected are those developed as membranous bones in connection with the primitive mouth, or stomodæum, *i.e.*, portions of the superior and inferior maxillæ.

Of the cranial bones the one most frequently affected was the frontal, which displayed bossing in six cases. In two more the parietal also showed enlargement, and in one (No. 29) the occipital showed a mesial boss. In three cases there was uniform unilateral enlargement of the cranial bones, without bossing. Thus in 11 cases out of 29 the osseous hypertrophy was hemicephalic rather than hemifacial.

Coming now to the cases in which the trunk and appendages shared in the overgrowth, in five of the ten observations the hypertrophy was unilateral, affecting the limbs or the limbs and trunk on the same side as the face (Nos. 11, 14, 16, 22, 28). In one (No. 20) the upper limb only, on the same side, was affected.

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[1] British Medical Association Meeting at Oxford, 1904. Discussion on Professor Cunningham's paper. Anatomical section, July 27.

In two the distribution was crossed, affecting either the face and arms on one side, and the leg on the other (No. 12), or the face on one side and half the body on the other (No. 26). In one (No. 24) the lower segments of the two upper extremities shared in the hyperplasia, along with half the face, and in one (No. 18) the facial hemihypertrophy was associated with acromegaly and giantism.

In addition to the cases already mentioned where the distribution of the hypertrophy in the appendages was segmental—*i.e.*, was limited by a boundary line, running at right angles to the long axis of the limb—the same feature was present in Case 26, where the thigh showed more marked enlargement than the leg and foot of the same side, and in Cases 21 and 29 where one hand, on the same side as the face, was enlarged in each case (cheiromegaly). A segmental distribution of the hypertrophy of the appendages was thus present in seven cases out of twenty-nine.

Unilateral enlargement of the tongue occurred in eleven cases. In six the corresponding tonsil was enlarged, and in five the teeth shared in the hypertrophy.

Cutaneous pigmentations, nævi or marbling of the skin, were present in seven cases. In one (No. 7) there was a patch of dark horny ichthyosis near the ear, and in one (No. 12) spots of vitiligo were scattered over the abdomen, on the same side as the hypertrophy, their distribution being bounded by the middle line.

Vaso-motor, or sympathetic, disturbances, such as hyperidrosis, excess of saliva, cutaneous hyperæmias, localised elevations of temperature, and excess of sebaceous secretion, are noted in seven cases (Nos. 2, 8, 9, 11, 12, 15 and 21).

Coarseness, or abnormal growth of hair, a trophic, probably, rather than a vaso-motor, symptom is reported in six cases (Nos. 2, 3, 8, 9, 13, 29).

As regards nerve function, motility and general sensibility are usually stated to be normal, but the latter was diminished in two cases (Nos. 12, 21). Subjective sensations of warmth, or a special sensitiveness to cold, with a cyanotic change of colour, under its influence is reported in five cases (Nos. 2, 12, 21, 16, 24).

Dissociation of sensation does not appear to have been looked for, unfortunately, in the majority of the recorded cases. In Lewin's case (No. 12) it was present.

The special senses were but seldom affected.

In two taste was blunted, and in one (No. 29) sight and hearing were damaged on the affected side. The defect of sight in this case was associated with neuro-retinitis, apparently due to pressure by osteophytic growth on the back of the ball, and the deafness was connected with chronic otorrhœa.

Prophthalmos was present in three cases (Nos. 8, 23, 29). The reflexes and electrical reactions were generally normal, and intelligence was usually unaffected.

In five cases the hypertrophy was progressive to a greater degree than the bodily growth (Nos. 1, 15, 17, 18, 29). In one (No. 26) it was retrogressive. This case, however, was an instance of the so-called chronic trophœdema.

Although, following the current nomenclature, the cases described here are distinguished as "congenital" and "acquired," it may be questioned whether the distinction is pathologically a valid one. That the condition is in the biological sense always acquired is evident by the fact that its hereditary transmission is unknown.

Even on clinical grounds it appears to be impossible—apart from the mere date of commencement—to separate between the two classes. Sabrazés and Cabannes are of opinion that a distinguishing feature may perhaps be found in the date of the dental evolution, which they consider to be delayed in the "acquired," and precocious in the "congenital, cases." But in the cases of Heath and Thomas (Nos. 19 and 26), which were "congenital," the dental evolution was retarded. Again, the unilateral enlargement of the tongue, which these authors consider to be usually absent in "acquired" cases, was present in that of Tilanus (No. 16), which was "acquired."

It does not appear possible, therefore, either on clinical or biological grounds, to maintain the distinction, and it would be more logical to consider the morbid conditions in both classes to be the outcome of some disease process common to them both, the date of commencement having been prenatal in one class and postnatal in the other.

PATHOLOGY.

Post-mortem observations of the condition may be said to be non-existent. In no case have the nerve centres been examined by modern methods.

Portions of soft tissues removed during life have shown as histological conditions, overgrowth of sebaceous and sudoriparous glands, and of adipose and connective tissues, with numerous degenerated epithelial cells.

The osteopathic changes have been well described by Mr. Jonathan Hutchinson and Dr. Alexis Thompson, and excellent illustrations of the conditions present in their specimens are given in Bramwell's "Atlas of Clinical Medicine," vol. III.

Both skulls show the over-development of bone to be limited strictly to the territory of the trigeminal.

Mr. Hutchinson describes the condition as one of overgrowth of bone, with broad-based exostoses. The overgrowth involves the frontal and sphenoid, as well as the facial, bones proper.

The hypertrophy in this case was said to have become conspicuous in middle life, but Mr. Hutchinson is of opinion that the case was probably congenital. Dr. Thompson's specimen was obtained from an insane patient who has been the subject of epilepsy since childhood. The overgrowth appeared to have commenced in early life, and not to have been congenital. It affected all the facial bones, and the anterior two-thirds of the cranium on the left side.

The convolutions on the anterior and superior aspect of the frontal lobe on the same side were atrophied by the pressure of a large exostosis projecting from the inner table of the frontal bone. To this Dr. Thompson attributes the epileptic seizures which occurred during life. A similar condition is described in the autopsy on Friedreich's case already referred to (No. 2), and in the second of the cases reported by Brissaud and Lereboullet (62).

PATHOGENY.

In the absence of pathological fact it is natural that speculation as to pathogeny should be exuberant.

The following theories are among those that have been put forward:—

(1) Vaso-motor.—The overgrowth being the result of vaso-motor paresis throughout the affected part, causing vascular dilatation and consequent hypernutrition and hypertrophy. With this view must be associated the theory of sympathetic irritation which has been favoured by several observers, *e.g.*, Barwell (20) and Hermanides (38).

The vaso-motor theory has the support of many writers, such as Trelat and Monod (5), Ziehl (10), Leblanc (41), and others, who base their opinions on the well-known experiments of Claude Bernard and Schiff (42), and on the observations of Stilling (14). In its favour may be cited the frequent vaso-motor derangements, vascular ectases, cutaneous hyperæmias, alterations in surface temperature, salivations, local sweatings, &c., which have been recorded in connection with the condition.

Both the vaso-motor and the sympathetic theories, however, only put the chief difficulty one step further back, as they do not explain why the vaso-motor paralysis, or sympathetic disturbance, arises.

Moreover, it must be noted that in many cases of facial hypertrophy, *e.g.*, those of Fischer, Dana, Montgomery, and my own, there has been a complete absence of vaso-motor indications of any kind. These considerations suggest that while the vaso-motor mechanism frequently shows signs of being implicated in the disease, it is probable that the implication is a secondary condition only, and that it occurs, when it is present, as the result of some deeper lying primary disturbance of the central nervous mechanism. A similar relation may be observed in the vaso-motor disturbances which accompany syringomyelia.

(2) The theory put forward by Fischer (9) of circulatory stasis in the affected part, produced by vicious positions of the fœtus in utero, and setting up hypernutrition and consequent overgrowth. Fischer supported his view by the observations of Miram (43) on the existence of hypertrophy in the distal parts of limbs in which congenital furrows were present.

This theory is obviously inadequate, inasmuch as it leaves the postnatal cases unexplained.

(3) Lannelongue's (44) view supposed some unknown stimulus or irritation of epiphysial cartilages and osteoplastic periosteum, which led to the overgrowth, first of bony, and secondly of soft, parts. This, too, must be put aside as insufficient to account for the cases in which the soft parts alone are affected.

(4) Messrs. Sabrazés and Cabannes (*loc. cit.*) have put forward a teratological theory, and consider the facial hemihypertrophy, when congenital, to be of the nature of an "anomaly by excess," which takes place in the frontal buds at an early date of intra-uterine life. They consider also that other factors may come into play, *e.g.*, variations in the size of the nutrient artery supplying the part.

This view, however, again fails to account for the "acquired" or postnatal cases, which, as we have already seen, appear to be not essentially separable from the congenital.

(5) The trophoneurotic theory. Lewin (13) appears to have been the first to put forward the clinical evidence in favour of a trophic, as distinct from a vaso-motor, theory, in accounting for local atrophies and hypertrophies.

The conflict which he mentions as existing then (1884), between the physiologists and the clinicians, regarding the existence of trophic influences generally, appears to be still unsettled.

Thus, Dr. Aldren Turner, in discussing the pathology of "Facial Hemiatrophy" (Allbutt's System, vol. vi., p. 488), dismisses the older views, including the "tropho-neurosis" theory of Romberg as scarcely worthy of consideration, while Mr. Jonathan Hutchinson, in the most recent discussion of the same subject ("Atlas of Illustrations of Clinical Medicine"—Fasciculus xix., 1904), after demonstrating that in a great majority of cases, if not in all, facial hemiatrophy is a late stage of fifth nerve morphœa, concludes that it is almost certain the primary sclerodermic changes are brought about under the influence of the nervous system, and that to them, therefore, the term tropho-neurosis is undoubtedly applicable.

The difference of opinion need not concern us here. Whatever the views of the physiologists may be regarding the conceptions of trophic centres and trophic influences, it is clear that the clinician and the pathologist, with the facts of syringomyelia and acromegaly before their eyes, cannot well do without them.

It may be well before proceeding further to recapitulate briefly the various forms in which hypertrophy of different parts of the body occurs.

As Anderson (45) has remarked, general, *i.e.*, complete bilateral, hypertrophy of the body is, at present, a possibility only. It may be considered that giantism, when free from taint of acromegaly, and when the bodily proportions are symmetrically preserved, would exemplify this class; but I am not aware of any authentic record of giantism which fulfils these conditions.

Complete unilateral hypertrophy, *i.e.*, of one half of the body, has of course been frequently recorded, as also has the partial unilateral type. In the latter form the face may or may not participate, and the hypertrophy may affect any limb, or segment of a limb, as in podo- and cheiro-megaly.

In the crossed variety of hemihypertrophy, various combinations may occur. The face on one side may be affected, with the trunk and limbs on the other, or the face and upper limb on one side, and the lower limb on the other.

The hemifacial variety of hypertrophy is thus seen to be merely a partial type, which may exist alone, or with which may co-exist any other form or degree of bodily overgrowth.

As regards the period of onset, whilst most instances in each class are congenital it is clear that all are not so. In Tilanus's case of complete unilateral hypertrophy the difference in size was not apparent till the child was 3 years old. In MacGregor's case (*Glasgow Medical Journal*, March, 1894) the partial hypertrophy apparently commenced in the leg at the age of 6, and in the hand at 10. In Montgomery's case, and in my own, photographs of the patients taken in early life show no existing facial asymmetry.

It will be noted that only in the hemifacial type does the area of hypertrophy correspond with the distribution area, complete or partial, of a peripheral nerve. In the bodily types, those, that is, affecting the trunk and limbs, the hypertrophied regions coincide with no area of nerve distribution, peripheral or radicular. The distribution of the hypertrophy is in these cases segmental or metameric, the boundary line, being, in the trunk, at right angles to the spine, and in the appendages, at right angles to the long axis of the limb. This apparent anomaly may perhaps be explained, as has already been suggested, by Gegenbauer's statement that the trigeminal, the nerve-area chiefly concerned in facial hemihypertrophy, represents the (unilateral) afferent fibres of the first cranial metamere.

It is in connection with this segmental distribution of the hypertrophies of the trunk and limbs, and, presumably, of the face as well, that Brissaud's theory of spinal and cerebral metamerism in nervous disease is of interest, through its conception of the cerebro-spinal axis as a series of neural metameres, or myelomeres, corresponding to and controlling as regards some nerve functions, *e.g.*, the trophic, the aboriginal metameric segments of the body.

Equally attractive as a theory, if less well founded on embryological fact, is the suggestion of outbuddings from the neural axis, in the brachial and crural regions, by which it is sought to explain (47) the distribution of those dystrophic conditións which affect segments of a limb only.

If now we enquire whether clinical observation warrants the supposition of a trophic influence inducing local overgrowth, and emanating from the central axis, it must be admitted that the facts of syringomyelia and of acromegaly support such a view.

While the dystrophies observed in connection with syringomyelia are most commonly of the atrophic type, it is well known that hypertrophic conditions have not infrequently been recorded.

Chauffard and Griffon (48) describe the case of a man of 48, who had observed for some years an augmentation in the volume of the right arm. Along with other signs of trophic disturbance (ichthyosis, hypertrichosis) the right upper limb was enlarged from the shoulder to the fingers, the bones being also affected. Dissociation of sensation was present in the whole length of the hypertrophied limb, and, in addition, in the shoulder, the right half of the neck, and right half of the cranium and face, the area of disturbance in the latter being limited accurately by the middle line. Perspiration was diminished over this area. No symptom of acromegaly was present unless the enlargement of the hand be so considered.

Somewhat similar cases have been described by Marie (49), in which, with syringomyelia there was present hypertrophy of the right arm and left foot, and by Charcot and Brissaud (50), in which enlargement of the left hand alone was present. Although designated "pseudo-acromegalic," the acromegalic symptoms in the above cases were limited to the enlargement of one or more extremities. In Lountz's case, however (51), there was hypertrophy of the right arm, including the bones, no suggestion of acromegaly being present, and a similar case is reported by Heldenberg (52) in which a man of 27, the subject of syringomyelia, developed hypertrophy of the right arm after an attack of acute rheumatism.

These cases show that there is sufficient clinical warrant for asserting an association between some forms of partial hypertrophy of the body, and lesions of the central gray matter of the neural axis.

The absence of hypertrophy of the cranial and facial bones in Chauffard and Griffon's case was remarked on by these authors, seeing that the dissociation symptoms extended to the cranium and face.

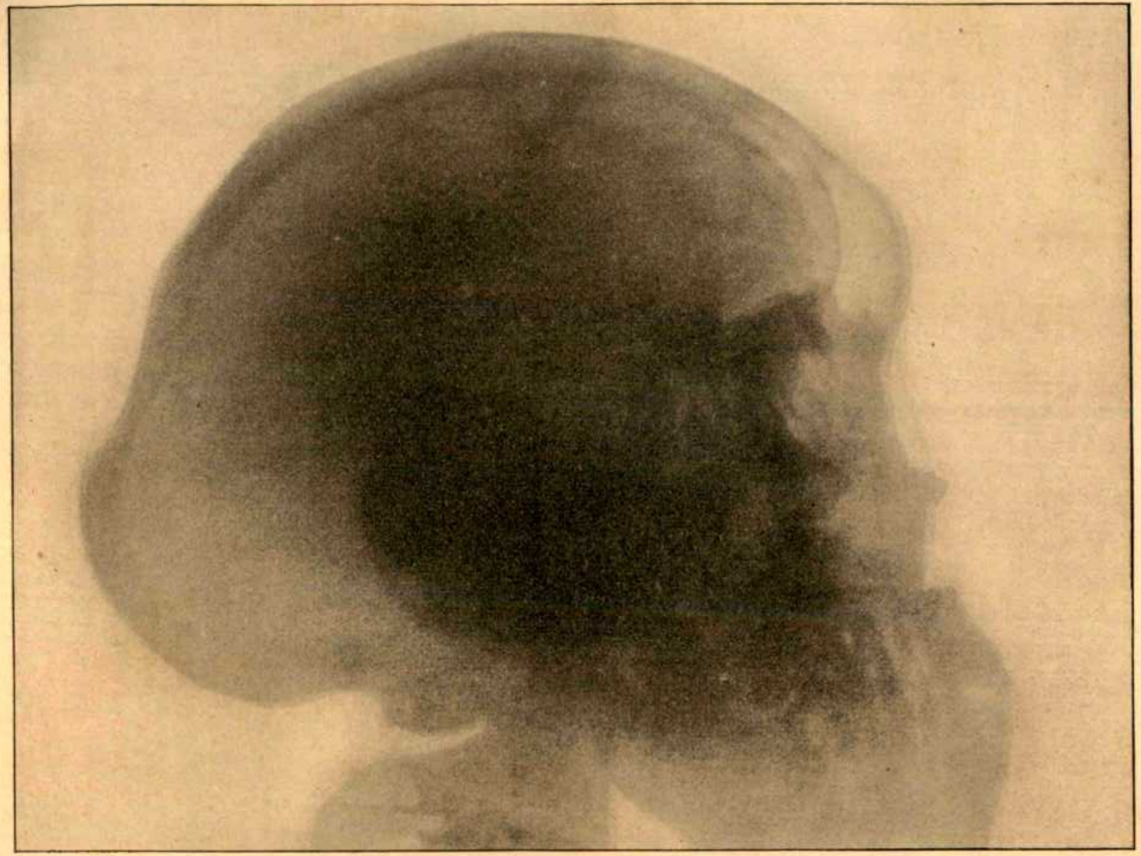

FIG. 5.

Radiogram of cranium of acromegalic giant, Charles F., to show "ressaut post-lambdoïdien." Slight facial hemihypertrophy was also present in this case.

(From Launois and Roy's monograph, by kind permission of Messrs. Masson et Cie.)

Hypertrophy of cranial bones, however, in cases of syringomyelia where no acromegaly was present, has been reported by Colman (53), in whose case frontal bossing occurred, by Westphal (54), who reported frontal and parietal thickening, and by Collier (55). In the latter case some enlargement of the parietal bone co-existed with remarkable deformity of the cranium.

It may be recalled that in 6 out of the 29 cases of facial hemihypertrophy here collected, frontal bossing was present, and that in two parietal thickening occurred as well.

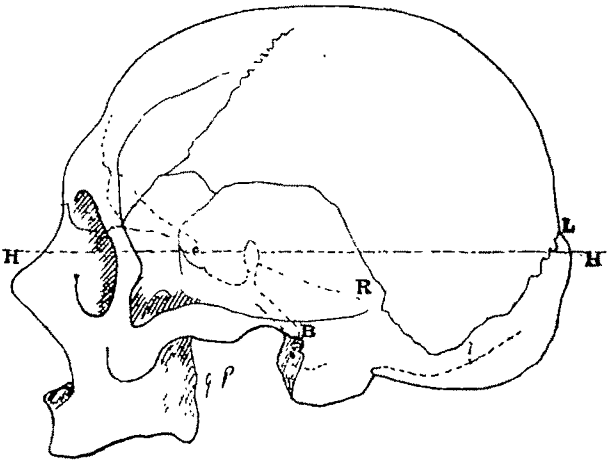

FIG. 6.

Acromegalic cranium in the Ecole d'Anthropologie, showing "ressaut post-lambdoidien" (Papillault).

(From "*Nouvelle Iconographie de la Salpêtrière*," vol. xv., p. 581, by kind permission of Messrs. Masson et Cie.)

The possible relationships between syringomyelia and acromegaly is a subject far outside the bounds of this communication, but in connection with the latter disease I wish to draw attention to the occipital boss reported in Case 29 of the present series. There is, so far as I am aware, no previous record of hyperostosis of the occipital bone in connection with facial hemihypertrophy. Its occurrence in this case appears to me to be of interest, as suggesting a possible link of relationship with acromegaly.

F. Schultze long ago called attention to the occasional prominence of the external occipital protuberance in this disease. Papillault later (56) described the so-called "*ressaut post-lambdoidien*," a bony overgrowth situated posterior to the lambdoid suture, the gradual development of which he had studied in the acromegalic giant Charles, and which he had also found present in an acromegalic skull.

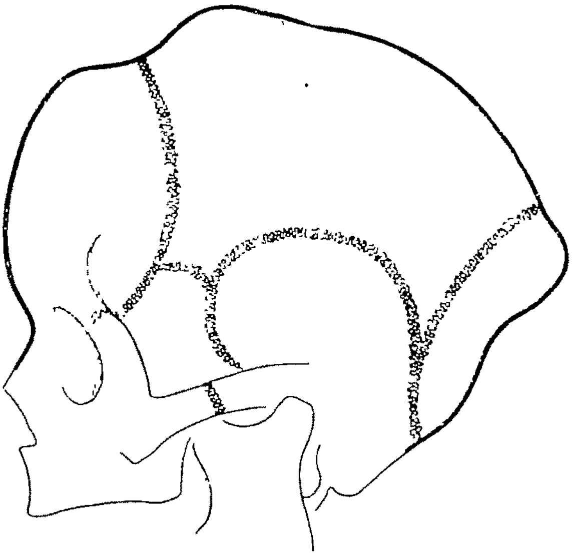

FIG. 7.

Tracing of skull in Case 29, taken by a strip of lead a little to left of mesial line, showing frontal, parietal, and occipital bossing (" ressaut post-lambdoidien "?) The thin lines and suture markings are diagrammatic.

From a comparison of a radiogram of the cranial bones in Papillault's case, with the condition present in Case 29, I am of opinion that the occipital boss in this case is of the same nature as that described by Papillault (see figs. 5, 6 & 7).

A further illustration of the relationship I have now ventured to suggest exists in Dana's case (No. 18), in which the two conditions of facial hemihypertrophy and acromegaly were present in the same individual. Another instance of the coincidence of these two conditions is recorded by Launois and Roy (64). In their account of the acromegalic giant, Charles F., these authors remark that facial asymmetry was present, the right half of the face being a little less developed than the left half. The patient himself had noticed that for the two months previously the left cheek projected more prominently than the right.

Dana remarks of his case that it might correctly be described as one of somato-megaly with hemiacromegaly of the head. Having regard to the frequency with which the cranial bones are affected, it would appear that some such term as hemicephalomegaly would express more accurately than facial hemihypertrophy the actual condition present, not only in Dana's case, but in many of the cases described in this paper.

The condition of chronic trophœdema, described by Meige in 1898 (57), is one that cannot be left out of account in future studies of facial and other hypertrophies.

Following Meige and his school, this may be described as a chronic enlargement, affecting, without prejudice to the health, one or more limbs, or segments of these, including it may be, one half of the face; presenting outwardly the appearance of an œdema, but characterised by absence of pitting, of pain, and of colour. Sensory troubles may be present, and in one case at least [Mabille (58)], dissociation of sensation has been observed.

The condition may be hereditary, or familial, congenital, or acquired.

Meige ascribes the condition to hyperplasia of the subcutaneous connective tissue, considers that it is related to the hypertrophic conditions met with in syringomyelia, and that it is due, pathologically, to changes in the trophic centres located in the central gray matter in the neighbourhood of the trophic centres for muscles. The local hyperplasia may not be limited to cellular tissue, that of fatty tissue may also occur. In the case of muscle, hypertrophy may also occur, though as a rule the muscular dystrophy takes the atrophic form.

As I have already suggested, it appears probable that some of the recorded cases of facial hemihypertrophy, those, namely, in which the soft parts alone have been affected, may have been conditions closely allied to, if not identical with those described as facial trophœdema by Rapin and Hertoghe (*vide supra*).

It is difficult in considering the pathogeny of acquired facial hemihypertrophy to avoid reference to the contrasted condition of acquired facial hemiatrophy.

Mr. Hutchinson's views upon this subject, founded upon Unna's account of the histology of the tissues in a case of scleroderma, have already been alluded to. Mr. Hutchinson is of opinion that the original local hyperplasia of subcutaneous fibrous tissue which is the essential change in the first stage of scleroderma, is of the nature of a trophoneurosis. The resultant arrest of growth in the lateral half of the face which constitutes facial hemiatrophy is thus to be assigned, if not directly, yet remotely and indirectly, to some disturbance of trophic, or nutritional, control akin to that which appears to underlie the changes in facial hemihypertrophy.

While the view that facial hemiatrophy is really an arrest of growth consequent on antecedent morphœa is doubtless true of all cases in which the atrophic changes have been observed before, or soon after, the cessation of period of adolescence, some doubt of the universal truth of this explanation will arise in connection with those cases in which the facial atrophy has commenced at mature age. Such are Bramwell's case of J. D. (59) in which the atrophic signs appeared first in a man of 40, and Hoffmann's case (60), where the first signs appeared in a woman, the subject of inveterate neuralgia, at the age of 42. In these cases the question of arrest of growth can hardly arise. Whether preceded by unobserved sclerodermic changes or not (and none were observed in either case), we have to recognise that at ages such as these there can only be retrogressive changes of formed adult tissues.

If we consider that in cases such as these we have pathologically, the converse condition to that which is present in facial hemihypertrophy, we may go further, and find a close parallel to the condition of acquired unilateral hypertrophy in the three remarkable cases described by Raymond and Siccard (61).

In two of these, concerning a brother and sister aged 30, and 35, respectively, progressive atrophy of one side of the body had been observed, extending from below upward, and involving in succession the leg, thigh, abdomen, thorax, upper extremity, shoulder, and one half of the face, the general health meanwhile remaining unaffected.

It is difficult to avoid in these cases, the conclusion arrived at by the authors themselves, that the condition is one of a trophoneurosis consequent on changes in the neural axis.

The following conclusions appear to be warranted by the considerations now put forward: (1) While nothing is known of the pathogeny of facial hemihypertrophy, the most reasonable explanation of the condition is that which associates it with disturbance of hypothetical nutritional or trophic centres, in the periependymal gray matter of the encephalon.

(2) In some of its features facial hemihypertrophy appears to have relationship with syringomyelia, and with acromegaly.

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Reviews.

Die Entwicklung des menschlichen Gehirns während der ersten Monate. Von Wilhelm His. (S. Hirzel, Leipsig, 1904. pp. 176).

The appearance of this monograph scarcely preceded its lamented author's death, and his precision and genius are so evident throughout it, that we cannot but regret it is the last work of one who has added so much to our knowledge of this branch of cerebral anatomy, who, in fact, almost created it.

His's apparent idleness during the past few years has been deliberate, as it has seemed to him advisable to allow the results of this fruitful period of the study of brain anatomy to accumulate, before concluding his work on its development.

As this volume only contains what has seemed to him definitely certain, it is incomplete in many respects, and is besides, in great part, a reiteration of his earlier publications, when he has been able to confirm or amplify the facts they contained.

His available material had increased to fifty-four embryoes, ranging in age from two weeks to the end of the fifth month, and to their study the same method of reconstruction from microscopical sections was employed. He commences with description of the histological development of the neural tube. The spindle-shaped spongioblasts, the elements of the myelospongium which have developed from the primary epithelium, are arranged radially, and by the syncytial confluence of the protoplasm of their two ends two concentric layers, the laminæ limitantes interna and externa are formed, while the nuclei of the cells lie chiefly in the intermediate zone. These three concentric zones are very apparent after the fourth week, and represent the first differentiation of the neural tube. Later, another layer comes into existence in the outer surface of the tube, the Randschleier, a protoplasmic net free from nuclei. The neuroglia develops only from the myelospongium through the intermediate stage of Deiter's cells; no mesoderm cells migrate into the neural tube as His formerly maintained. The neuroblasts which begin to appear on the border of the membrana limitans interna arise by the unilateral outgrowth of the protoplasm of germ cells, whose nuclei divide by mitosis. Distinction between these and the spongioblasts is at first very difficult, and can only be definitely determined by their connections with nerve-fibres. All nerve cells, no matter how complex their later arrangement, have their origin in the central portion of the neural tube, and the organisation of the system is dependent on the migration of its cells, and on the arrangement of the fibres which spring from them. The former may remain at the site of their origin, as in the spinal cord; they may migrate to the surface, as those of the cortex of the cerebral hemispheres; or may otherwise change their position in the matrix, as do the cells of the inferior olives and of the pontine ganglia.

All his recent observations support the author's original contention of the origin of each nerve-fibre as the process of a single cell. He argues against Bethe that the origin of fibrils from the cells of the sheath of Schwann has not been definitely proved, free from objection, as there is much room for the misinterpretation of the histological appearances. Further, in mammals the sheaths of Schwann and their nuclei appear relatively later than in the lower vertebrates, on the study of whom Bethe's statements are based, and in man the bundles of nerve fibres are distinct before their appearance. Further, Bethe's observations cannot be applied to the central nervous system.

The development of the ventral portion of the neural tube precedes that of the dorsal. During the first month of embryonic life nerve fibres begin to develop, the first being those of the ventral spinal roots, but soon later the sensory fibres, and, almost simultaneously, those of the acusticus appear. The olfactory fibres can only be seen some time later, and the optic are the last to develop.

The first intramedullary bundles which can be made out are those of the ventral columns. These are chiefly composed of arcuate fibres springing from cells in the dorsal portion of the cord, which enter them directly, or after having decussated in the ventral commissure, but, in addition, cells lying in the ventral portion of the cord give origin to fibres which run longitudinally. At this time the dorsal columns only contain the fibres of the neighbouring roots, and their further development is chiefly due to the longitudinal growth of these.

The metencephalon undergoes analogous changes, but at a somewhat later period. A ventral commissure of arcuate fibres, similar to that of the cord, which extends from the cervical region to the isthmus, is developed early. So also are the fibres of the dorsal longitudinal bundles, each of which is probably a complex of shorter tracts, which extends caudalwards into the ventral columns of the cord. In the first month, while there are scarcely any neuroblasts in the dorsal portions of the mid-brain or hemispheres, a continuous chain of them extends along the border of the hypothalamus from the optic vesicles to the mid-brain.

The development of the fore-brain is more complicated. The hemispheres appear early, even before the closure of the neuroporus, as rounded evaginations. The rhinencephalon develops basal to the neuroporus, and so cannot at any stage be confused with the pallium, and the corpus striatum from a depressed triangular area ventral and rostral to the hemispheres, and dorsal to the hypothalamus. In the beginning of the second month the primary longitudinal fissure rapidly deepens, and separates off the lateral ventricles from the primarily single cavity. The hemispheres also increase in size and begin to project over the 'tween-brain, and the rhinencephalon is definitely separated from the thalamus.

His joins in the discussion as to the nature of the so-called transitory furrows of the hemispheres, and comes to the conclusion that they are only the result of *post-mortem* shrinkage. The only definite indication of the existence of a true furrow is the thickening of the soft membranes opposite it, or their projection into it.

The hemispheres develop rapidly from the second month till, in the fourth, they have attained almost their normal form. The development proceeds from the region of fusion of the pallium and corpus striatum toward the apex, and, last, to the mesial surface. It is there that the first germ cells, which, by mitotic division, produce the future cortical cells, are found in the inner layer of the hemisphere. The pyramidal cells slowly migrate from the ventricular surface through the thickness of the hemisphere wall into the cortical layer, the first reaching it in about the eighth week. They are generally bipolar structures, with the longer and more definite process directed centralwards. They further differentiate and change their form as they pass out, and accumulate till the cortical zone contains several layers of them. They first appear in the mesial surface of the hemisphere in the third month, but that portion of it and the caudal end of the rhinencephalon where the two hemispheres later fuse and from which the corpus callosum and fornix develop, remain free from cortex.

In the second month the first fore-brain bundles appear; these, which are the thalamic radiations to the pallium, come from the region of the internal capsule, and break up in the lateral surface of the hemisphere. The steady increase in thickness of the hemisphere walls is chiefly due to growth of its intermediate layer (Zwischenschicht), and it is later at the expense of this that the convolution of its surface is effected.

The corpus striatum develops *pari passu* with the pallium. Its anterior end is early divided into three limbs by the Fissura epirhinica, F. mesorhinica, and F. metarhinica. The anterior limb fuses with the lateral wall of the anterior olfactory lobe, and the posterior with the mesial wall of the hemisphere, their union being the structure through which the fornix passes on its way to the corpora mamillaria. The ventro-caudal end of the nucleus caudatus develops considerably later. Then, as the hemisphere bends over the 'tween-brain, the nucleus caudatus curves into semi-lunar shape, with its anterior end resting on the rhinencephalon and its caudal in the inferior horns of the lateral ventricle. The appearance of the internal capsule in the third month divides off the nucleus lenticularis. Embryologically considered, this, with the claustrum, is a portion of abortive cortex rudiment.

From the end of the second month the pallium exceeds the rhinencephalon in size. This is divided into a rostral and a caudal portion by the F. mesorhinica; from the former the olfactory bulb and trigenum develop, and the latter forms the trapezoid area (Trapezfeld) from which the central connections of the olfactory system are developed, and which is of considerable importance in the secondary fusion of the hemispheres. The anterior commissure results from fibres which grow through the secondary fusion of the two trapezoid areas; it first appears in the eleventh week, and its development is complete in the middle of the third month. Similarly, the corpus callosum and fornix develop from the fusion of the limbus areas of the mesial walls of the hemispheres. At first only the middle portion of the former appears, the genu and splenium being of later formation.

The development of the cerebellum begins later than that of the hemispheres. At the end of the second month a prominence appears on each side of the metencephalon, and these two, having increased in size, fuse medially. The earliest portion of the corpora restiformia only contains olivo-cerebellar fibres, whose termination in the vermis is very easily followed.

A concise table shows the dates of the successive appearances of the intramedullary tracts, the spinal root of the trigeminus, the fasciculus solitarius, and the dorsal longitudinal bundle being among the first to appear, and the cortico-spinal and pontine bundles the last.

The monograph is so replete with facts, newly discovered or authoritatively confirmed, that an adequate summary of its contents would require more space than is here available.

Only one feature offers itself to adverse criticism. The volume is liberally supplied with illustrations, but the majority of these are photographs, often of sections under high magnification, which leave much to be desired in distinctness. They have evidently been deliberately preferred to drawings or diagrams, that the author may represent exactly what he sees; but he has been able to examine them under conditions very different to those of his readers, and they, on the other hand, are ever ready to assume so much scientific honesty on his part as to allow him to temper exactness with clearness. The lack of any lettering on the illustrations often gives considerable trouble in exactly determining what is described, and frequently necessitates repeated reading.

GORDON HOLMES.

Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete des Neurologie und Psychiatrie. VI. Jahrgang. Bericht über das Jahr, 1902. (Published by S. Karger. Berlin, 1903. Price 35 marks.)

THE sixth volume of this Jahresbericht contains a catalogue of all the papers published during the year 1902 on Neurological and Psychological subjects. Not only is it a catalogue, but it also contains a digest of the most important papers on the various subjects. To all those who are engaged in looking up the recent literature of a subject this work is invaluable, for, in many instances, when from the title of the paper it might be considered necessary to look up the original article, the reference to the digest of the paper will show whether it has bearing on the subject under consideration or not. How completely each subject is reviewed may be judged from one instance taken at random, viz., the digest on "brain tumours," by Dr. Bruns, for, out of some 135 references on this subject, no less than sixty-two papers are abstracted. The uniformity of arrangement of the subjects is of the greatest value, for it is a simple matter to find out all the papers published within the last six years on a given subject by referring to the heading in the present and past volumes.

FREDERICK E. BATTEN.

Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete des Neurologie und Psychiatrie. VII. Jahrgang. Bericht über das Jahr, 1903. (Parts I. and II. Published by S. Karger, Berlin, 1904. Price 35 marks.)

THE digest of the various papers published during the year 1903 on Neurological and Psychological subjects has been divided into two parts. No alteration has been made in the arrangement of the subjects dealt with, and the two parts complete the digest of all papers on Neurological and Psychological subjects published in the year 1903. The Editor and the publisher are indeed to be congratulated in that they have succeeded in producing this digest within such a comparatively short period after the publication of the papers reviewed therein.

Like its predecessors this volume is a monument of systematic and careful work, and for the purpose of acquiring a complete knowledge of all the recent work on a given subject, there is no review or digest to compare with the above.

FREDERICK E. BATTEN.

Technique de Psychologie Expérimentale. By Dr. TOULOUSE, N. VASCHIDE et H. PIÉRON. Bibliothèque internat. de Psychologie expérimentale, 1904. (Paris: Octave Doin. Pp. 335. Prix 4 fr.).

IN this book Dr. Toulouse and his collaborators at the Ecole des Hautes Etudes claim to have elaborated a system of tests adapted for the psychometric study of individuals. Those who have read Dr. Toulouse's earlier volume, embodying his interesting experimental researches on the novelist Zola, will be familiar with the general scheme of his present undertaking. The authors advisedly lay stress on the essentially tentative character of the technique proposed; and it would be idle to ignore the defects and errors which are inevitable in pioneer work of this kind. One can but hope that it may pave the way to the ultimate establishment of an internationally recognised system of psychometric tests, analogous to the anthropometric standards of the Frankfort agreement.

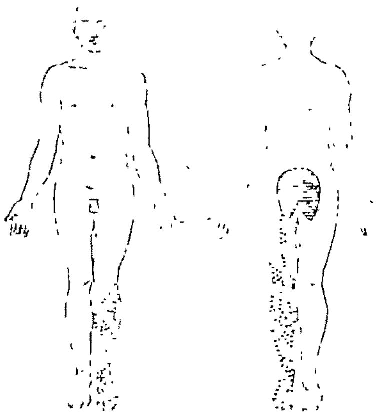
C. S. Myers.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

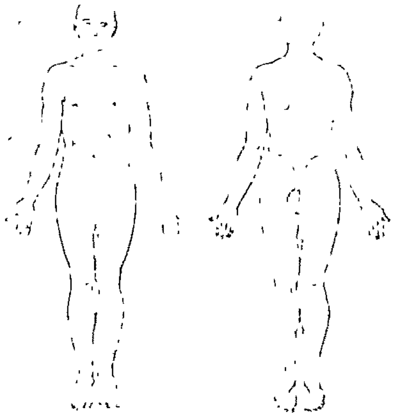
At the Clinical meeting, held at 11, Chandos Street, on Thursday, October 27, at 8.30 p.m. (patients in the room at 8 p.m.), the following cases were shown:—

Dr. **Ferrier** and Mr. **Ballance**.—*Case of recovery after operation for cauda equina lesion.*

Mr. C. H., solicitor, aged 41. Patient came under observation in February, 1903, with the following history:—Two attacks of pleurisy with effusion, nine and four years ago respectively. Two attacks of hæmoptysis within last two years. Four years ago, when wrestling, he fell on lower part of back and since then has complained of intermittent pains in outer side of left thigh. Six weeks ago pain became worse. Four weeks ago pain appeared in right thigh also, the left lower limb became paralysed



C. H.—Anæsthesia, February 6, 1903.



C. H.—Anæsthesia, October 14, 1904.

below the knee, and a week later the right also. With the appearance of motor weakness, there developed anæsthesia of perineum and left lower limb and anæsthetic incontinence of urine and fæces. On examination there was no tenderness or irregularity of the spine and only a doubtful bulging over upper sacral region. There was moderate anæsthesia to all forms of stimuli on the left side from L 4. root downwards, and on both sides, more intense, from S 3. root downwards. Movements of left hip and knee were weak. Below the knees there was flaccid paralysis of all muscles on both sides, except feeble flexion of right hallux. Knee jerks, right faint, left absent, ankle jerks absent. Cremasteric reflexes brisk. Bulbo-cavernosus reflex absent, superficial and deep anal reflexes absent. Anæsthetic incontinence of urine and fæces. No bed-sores, but two days before operation there appeared a small area of reddened skin to the left side of the middle line over the sacrum. Occasional partial priapism.

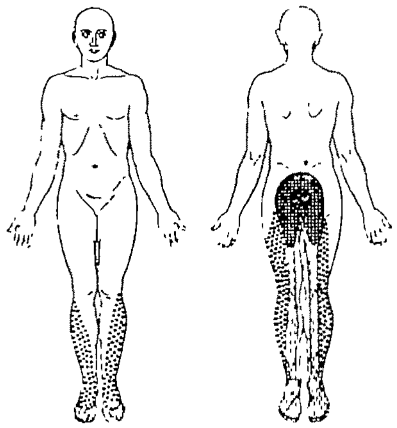
Operation by Mr. Ballance on February 20, 1903. On removing the laminæ of the three lowest lumbar vertebræ, thick tuberculous pus was found, outside the theca, chiefly in front of it. A small cavity was found in the body of the fourth lumbar vertebra.

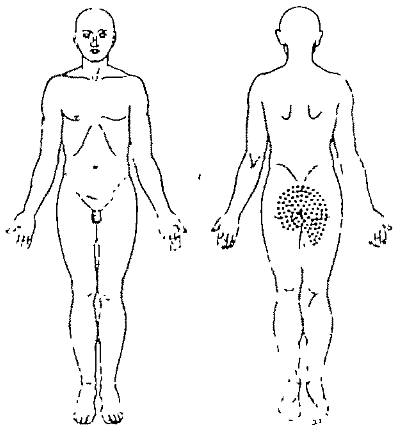
Present condition. October, 1904.—Patient has recovered all movements of both lower limbs, and can walk four miles without fatigue. Gait normal. There is still an area of anæthesia to the left of the middle line, corresponding to the fourth and fifth sacral and the coccygeal root. The anæsthesia does not include the genitals, but affects the left wall of the anal canal. Knee-jerks brisk and equal. Ankle-jerks, left present; right, absent. Plantars flexor. Anal reflexes, superficial and deep, absent, also bulbo-cavernosus reflex. Bladder normal. Imperfect control over rectum if motions are loose. Bowels tend to be confined.

Dr. FERRIER and Sir V. HORSLEY.—*Case of recovery after operation for tumour of cauda equina.*

S. C., a boy now aged 14, was operated on by Sir V. Horsley, at the National Hospital, on October 27, 1903. The history prior to operation was as follows:—For twelve months he has had constant dull pain in the right thigh, worse on exertion, and causing him to walk badly. A month ago the gait became much feebler, with difficulty in raising feet from ground, the left foot being first affected, but latterly the right being the feebler. For six months there has been some delay in micturition, with occasional incontinence. For a month there has been a numb sensation in the soles of both feet, and some loss of sensation observed by the patient in the buttocks.

On examination there was impairment to all forms of sensation in both lower limbs from the fifth lumbar root downwards, symmetrical on the two sides. The anæsthesia increased in intensity from the upper roots downwards, being absolute in the lower sacral root areas. There were wasting and weakness, with diminished faradic reactions, in all the muscles of the left lower limb, and similar weakness, with diminished reaction in the right anterior tibial group. The ankle jerks were absent, so also was the left knee jerk. The right knee jerk was only obtainable on reinforcement. The plantar reflexes were absent. There was delay in micturition, with occasional incontinence. The anal sphincter was normal. There was no spinal deformity.


S. C.—Anæsthesia, October 20, 1903.


S. C.—Anæsthesia, October 17, 1904.

Sir V. Horsley removed the upper two lumbar and lowest two dorsal spines and laminæ. The theca was seen to be distended in the lower part of the wound, and markedly constricted towards the upper part. On incising the dura, a grayish tumour, 5½ × 3 cm. (microscopically a fibro-myxoma) was exposed, enveloping the cord posteriorly and laterally, not infiltrating it, and apparently arising from the fourth lumbar posterior root on the right side. The tumour was entirely removed, and the cord was seen to be considerably compressed at this level.

Patient rapidly recovered motor power, and left hospital in January, 1904. Subsequently a dorso-lumbar kyphosis developed, for which he has had to wear a spinal support.

Present condition.—He can walk well, without any abnormality of gait. Occasional slight delay in micturition. No incontinence. Area of anæsthesia limited to lower sacral and coccygeal root-areas, nowhere absolute. Knee jerks, brisk and equal. Ankle jerks, left greater than right. Plantars flexor, cremasters brisk. Bulbo-cavernosus reflex normal.

Dr. Colman.—*Case of myasthenia gravis with prominent bulbar symptoms.*

L. M., aged 23. Has had rheumatism twice, and has a slight systolic murmur, but no cardiac symptoms. Subject to sore throats, but has never had diphtheria. Nine months ago noticed that her lips and tongue became fatigued by talking, and that her voice became husky. She also found that her arms and legs easily became tired, the fatigue passing off after a short rest.

On admission to hospital she was well nourished. There was obvious constant weakness, even when at rest, of the levator palpebræ superioris and the orbicularis oris, the lower lip hanging down somewhat. Voice muffled and somewhat nasal. No obvious weakness, when at rest, in limbs. All muscles show rapid "fatiguability." After reading aloud for a quarter of an hour the lower lip becomes pendulous, and the palate almost immobile. Similarly at the end of a meal she has difficulty in preventing food escaping from the lips, and she has nasal regurgitation. These symptoms rapidly pass off if she takes a short rest. Movement of the arms and legs as in knitting and walking quickly causes fatigue, and she has to take frequent rests. *Sensory symptoms.*—When the muscles are fatigued by exertion, she complains of an aching sensation in the muscles concerned, and of tingling in the lips, palate or limbs. Beyond some doubtful anæsthesia of the palate, no loss of sensation to any forms has been actually demonstrated. *Electrical reactions.*—The muscles react readily to both faradism and galvanism, but are rapidly exhausted by repeated weak faradic stimuli ceasing to react to isolated shocks in two minutes, but quickly recovering after rest. It was found that tetanisation did not produce fatigue as quickly as a succession of isolated shocks.

Dr. ALBERT WILSON.—*Progressive muscular atrophy with arthritic changes.*

H. H., aged 25, in perfect health and an athlete until July, 1898, when he injured his right ankle entering a railway carriage. He was lame for two years, and the foot became everted, when it was found that the inner malleolus was broken off (skiagrams). A boot was constructed to rectify this, but a slip off the kerb bruised the ankle, and it began to swell and caused pain and became useless.

The swelling of and pain in the bone continued, and his general health was affected. Cough came on and there were signs of incipient phthisis at the left apex, with moist crepitations.

In May, 1902, I amputated the right foot (Syme). The section of the enlarged tibia was very vascular and the bone soft.

Patient went to Margate for a month, apparently relieved. Here he contracted 'Rheumatism' and was confined to bed with swelling of the left wrist and right elbow and pain in the left knee.

About October or November gradual muscular wasting commenced.

It began in the right biceps and triceps, then in the left forearm, following the swollen joints.

Then the wasting became general and rapid and all his joints seemed to stiffen. Even the jaws became stiff.

Present condition.—The body is devoid of all subcutaneous fat.

Facial muscle of expression active, and the patient can whistle.

The jaws will only separate one inch, and any attempt to force the lower jaw open causes pain in the joints.

The joints of the atlas and axis are anchylosed and much enlarged laterally.

The head is fixed and immovable. The neck muscles are rigid.

The right longissimus dorsi and right trunk muscles are atrophied, so also the supra and infraspinati on both sides.

The right serratus is atrophied, the left is about normal.

Both pectorals, deltoids, triceps, and biceps, are extremely atrophied.

The right elbow joint is swollen and movement limited to half flexion.

Both hands are semiprone and pronate easily, but cannot supinate.

The forearm and hand muscles are much wasted.

Interossei and lumbricales about normal.

The left wrist is swollen and dislocated.

The left carpus is pulled over on the flexor side.

The metacarpals are pulled on to the extensor side.

The opponens is weak. He can move the fingers.

The glutei are extremely atrophied, leaving concavities.

The vasti interni are atrophied, so also the calf muscles.

The left knee is swollen with special enlargement of the internal condyle and extension limited to half the normal.

The left foot and ankle are swollen and œdematous, so that there is no lateral movement.

There is slight anæsthesia in the legs and arms. No analgesia. All the reflexes are much exaggerated, a tap on the knee causing sudden and painful spasm of the whole limb and trunk.

Mr. CHARLES BALLANCE.—*A case of facial palsy treated by hypoglosso-facial and gustatorio-hypoglossal anastomosis.*

J. F., aged 7 years, was admitted to St. Thomas's Hospital last June. Since early infancy he had had purulent discharge from the left ear, and complete left facial palsy. On several occasions glandular tubercular abscesses had been opened in the neck.

June 18.—Removal of temporal bone disease.

June 29.—Hypoglosso-facial and gustatorio-hypoglossal anastomoses carried out. During this operation several caseous glands had to be removed. The facial nerve was found imbedded in scar tissue in the lower part of the parotid gland, and completely severed by old suppuration. On the day after operation, distinct voluntary movement of the left angle of the mouth occurred. This disappeared in a fortnight, and complete atrophy of the left half of the tongue took place.

Present condition.—Commencing movement of the angle of the mouth is present, and the left half of the tongue is much less atrophied than formerly.

Mr. CHARLES BALLANCE.—*A case of facio-accessory anastomosis for facial palsy, with recovery of dissociated movement.*

M. K., a girl, aged 18 years, was admitted to St. Thomas's Hospital in 1902, for facial palsy due to an operation performed for ear disease at another hospital some seven months previously.

Operation. — August 1902. — Facio-accessory anastomosis. Five months later no recovery had taken place. Patient then disappeared, but was seen eighteen months after the operation. Recovery had then taken place, so that without movement of the shoulder, the previously paralysed face moved voluntarily and emotionally in symmetry with the other side.

Mr. HAMILTON BALLANCE.—*A case of facio-accessory anastomosis for facial palsy.*

A married woman, aged 33, was operated on for chronic right otitis media at the Norwich Hospital in 1901. This was followed by facial palsy. No signs of improvement in six and a half months taking place, facio-accessory anastomosis was performed.

The result has been that the muscles all respond to faradism, and at rest the face is symmetrical, but no movement of the face occurs without movement of the sterno-mastoid and trapezius. During emotional movement of the face the deformity is as great as before the operation.

Dr. PURVES STEWART and Mr. LENTHAL CHEATLE.—*Case of facio-hypoglossal anastomosis.*

J. J., a young woman, aged 26, who had been deaf since childhood from chronic middle ear disease, was operated on six and a half months ago for right-sided mastoid disease. During the operation the facial nerve was divided. Total facial palsy resulted, with loss of taste in the anterior two-thirds of the tongue. A fortnight later, *i.e.*, six months ago, the patient was seen by Mr. Cheatle, who performed end-to-end facio-hypoglossal anastomosis. Two months ago slight voluntary power reappeared in the lower face, so that on pushing the tongue into the left cheek the right angle of the mouth was drawn outwards.

Present condition.—There is hemiatrophy of the tongue, owing to the hypoglossal palsy. The mouth at rest no longer droops on the affected side, and the patient has fair power of movement in the lower facial muscles.

Particular attention is directed to the fact that facial movement occurs not merely in association with tongue movements, but that there is *dissociated* emotional movement, *e.g.*, on smiling.

The upper facial movements have not yet recovered.

Mr. Charles Ballance.—*A case of temporo-sphenoidal abscess with loss of the sense of smell.*

T. M., aged 41, was admitted to St. Thomas's Hospital on October 6, 1903.

History.—Discharge from right ear since early childhood. Twelve months ago son died of cerebral abscess.

Present illness.—Severe headache for one week, chiefly frontal. Yesterday he vomited once. He volunteered the information that for a week he had lost all sense of smell.

Signs and symptoms.—Foul pus in right auditory meatus. Both pupils moderately dilated. Right pupil stabile as compared with left. Slight right optic neuritis. No paralysis of face or limbs. No anæsthesia. Reflexes normal.

Operation.—October 6, 1903. After the completion of the mastoid operation a little pus was visible oozing from the tegmen antri. The tegmen was removed and the stalk of the abscess adherent to it was used for the passage of a drainage tube. Two ounces of fetid pus with gas escaped.

Recovery was rapid and uninterrupted. No hernia occurred. The sense of smell has returned.

Dr. Beevor.—*Pseudo-bulbar paralysis from bilateral hemiplegia.*

S. M., aged 23, soldier, had specific disease in 1900, otherwise healthy, family history unimportant.

In July, 1903, suddenly without loss of consciousness, lost power in *left* face, arm and leg; unable to open mouth for two weeks; speech indistinct. In November, 1903, during sleep, lost power suddenly in the *right* arm and leg, (?) face. Speech was slightly affected. He improved so as to walk and talk fairly well. In June, 1904, had third attack, and was admitted into the Great Northern Hospital, not unconscious, lost power in *left* arm and leg, and face, and control over sphincters lost; speech completely lost, unable to phonate, but could groan; could not swallow; had paralysis of soft palate; could not open or shut mouth, bite, or move the tongue; he could not elevate or retract angles of mouth, purse up mouth, or close eyelids, he could allow them to fall over the eyeballs, but was unable to corrugate the brows. The movements of the eyes normal in all directions, and in looking upwards forehead wrinkled by frontalis. He could not voluntarily inspire or expire, and could not voluntarily cough, though he could do so reflexly. He was very emotional, and always laughing or crying, and in retraction of angles of the mouth in laughing, the left side moved later and less than right, but in yawning both sides moved equally. The left arm and leg were slightly weaker than the right, but he could perform all movements. The left latissimus dorsi acted well as a unilateral adductor of the left humerus, but in the voluntary bilateral movement of coughing, or even of expiration, there was no movement of either latissimus dorsi, though the muscles of both sides contracted equally in emotional laughing and in reflex coughing. The knee jerks were increased but L. > R., there was ankle clonus on both sides. Plantar reflex gave extension of left big toe and flexion of right. In about two weeks time he could just close the teeth, but he had still to be fed by a nasal tube. In another ten days he could open the mouth, and protrude tongue just beyond the teeth. In ordinary respiration the right side of the chest moved more and sooner than the left, and the same observed in forced breathing, which he could now to a slight degree perform (the opposite to what occurs in hemiplegia). On admission into the National Hospital, on July 29th, he could just pronounce some of the vowel sounds, he could swallow with difficulty, and saliva dribbled from mouth. He could not voluntarily perform defæcation and he had precipitate micturition. He has gradually improved, and can now close the eyes and show his teeth, and can just purse up mouth, can inspire and expire and faintly cough; in ordinary respiration the movements of the two sides are equal, in voluntary inspiration, R. > L. The lesion is considered to be bilateral, due to specific thrombosis of the branches of the posterior communicating arteries to the genu of the internal capsule of both sides, and the great interest of the case is the paralysis of many voluntary bilateral movements about the face and fauces and of respiration with preservation of the emotional and reflex movements, and also with only slight weakness of the voluntary movements of the limbs.

Dr. RISIEN RUSSELL.—*Case of pseudo-bulbar paralylis (double hemiplegia).*

E. G., female, aged 24 years, single. Attack of left hemiplegia in 1896, from which she recovered. Similar attack on the right side eighteen months later, but attended with inability to speak, difficulty in swallowing, with choking and regurgitation of liquids through the nose, and dribbling from the mouth. Recovery of power in the limbs, but only slight improvement in the bulbar symptoms.

Emotional display in excess. Difficulty in sustaining ocular movements. Movements of jaw slow, spastic and weak. Volitional movements of face weak and laboured. Inability to blow out cheeks or whistle. Excess of expressional movements of face in smiling. Associated movements of face as in firmly grasping an object practically lost. Scarcely any movement of palate on phonation. Movement good in swallowing. Considerable but slow movement on reflex stimulation. Almost complete anarthria. Deglutition difficult and slow. No regurgitation through nose, but liquids dribble from mouth. The tongue is not wasted, but cannot be protruded to any extent nor moved from side to side. Muscles of neck and trunk act well, but latissimus dorsi contract relatively less on coughing than on voluntary depression of the arm. Slight uniform weakness of the limbs, more marked on the right side. No rigidity. No ankle clonus. Plantar reflex flexor in type. Sphincters unaffected.

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EDITOR.

BRAIN.

PART IV., 1904.

Original Articles and Clinical Cases.

ON THE SPINAL CORD DEGENERATIONS IN ANÆMIA.

BY J. MICHELL CLARKE, M.A., M.D., F.R.C.P.

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THE exact relationship of anæmia to the degenerations in the spinal cord which are comprised under the various terms, "diffuse degeneration," "combined sclerosis," and "subacute combined sclerosis" of the cord, is still a matter about which there is considerable difference of opinion. From a clinical standpoint, anæmia may be associated with degeneration of the spinal cord in at least three ways: (1) it may distinctly precede the spinal changes for a longer or shorter time; (2) it may accompany them from the first, and proceed *pari passu* with them; or (3) it may appear late in the course of the cord affection, just as it often occurs in the later stages of any chronic illness. In the last instance, the anæmia is not as a rule so profound as in the other two. It should be added that in any case the anæmia may from time to time undergo considerable remissions, [illegible]onally be cured.

Although the subject is not a n[illegible]y cases having been published, as it is stil[illegible] extent *sub judice*, the following cases, which belong entirely to the first group, where anæmia predominates over and precedes the spinal symptoms, and which illustrate both the early stages of the degeneration in the posterior columns of the cord and also their later development, have seemed to me not unworthy of record. I wish to emphasise that this paper deals only with cases in which the cord changes were, so far as one could judge, secondary to antecedent severe anæmia. In two of them disease of the cord was not suspected during life, and in all the anæmia was of considerable duration. It is not, therefore, a question in these cases of anæmia appearing in the course of primary chronic spinal cord disease. The second case is of especial interest, as I was enabled to watch it throughout the whole course of the illness; it presents the cord affection in an early stage, and is further interesting in that the anæmia was cured, and the cord changes had apparently ceased to advance.

With regard to the form of anæmia present in these and similar instances it is now established that it is by no means only, nor even most often, pernicious anæmia. In the published cases there is a lack of exact observations on the blood, and I regret that this fault unavoidably attaches to one of two of the following reports.

The first case was the first example in which the slighter changes in the cord came under my observation, and appeared to represent the earliest stage in the cord affection of anæmia, but as the patient was only under observation during a short and fatal illness this was not certain. The correspondence of the lesion, though in a less advanced form, with that in Case 2, in which, as will be seen, there is no doubt that the degeneration is in the early stage, proves, however, that this first case shows the commencement of the cord lesion of anæmia.

Case 1.—The patient was a man, aged 26, who was admitted into the [illegible] General Hospital, under Dr. Harrison, for pneumo[illegible] a few days later. He was profoundly anæmic [illegible] of the blood gave red cells 660,000 to c.m., irregula[illegible] shape; hemoglobin, 16 per cent.; blood decimal [illegible]

Besides the ordinary lesions of pneumonia and general anæmia of all the organs the chief changes found *post mortem* were as follows. The non-consolidated areas of the lungs were rusty in colour; the heart small, but not fatty; the pancreas somewhat indurated; the mucous membrane of the small intestines thin, and in parts atrophic; Peyer's patches inconspicuous; the intestinal mucosa nowhere gave a pink colour with mercuric chloride solution; no iron reaction in liver, spleen, or kidney.

The brain and spinal cord were apparently healthy, except that they were very anæmic.

Microscopic examination of the organs showed no noteworthy changes, except in the spinal cord. Sections were made of all parts of the cord, and exhibited partial degeneration of the posterior columns, entirely confined to the cervical portion, and not quite extending to the lower limit of that region.

The extent of the degeneration is shown in figs. 1 and 2. It was most extensive at the level of the third and fourth cervical roots, occupying the greater portion of the central parts of the posterior columns, not affecting, however, the anterior and posterior extremities of the column of Goll, and leaving an unaffected zone of varying width on the dorsal surface of the cord, and lining the posterior commissure and posterior cornua. At the level of the fifth and sixth cervical the degeneration formed on each side a narrow t[illegible]ncated area, the broad end dorsally, in the lateral border of the posterior median, and mesial part of the postero-external, columns. The posterior roots were normal, and there was no evidence of the entrance of degenerated fibres from them into the cord. The sections stained by Marchi's method showed a few scattered degenerated fibres in those parts of the posterior columns which, by the Pal-Weigert stain, appeared healthy, and also a very few in the lateral columns; both sets in the cervical region only.

The gray matter and other parts of the cord were normal.

Case 2.—Profound anæmia, three and a half years' duration; paræsthesiæ and paresis of legs after two years; recovery from anæmia; death from gangrene of lung following pneumonia; post mortem, stationary changes in cord.

J. S., aged 28, gardener. First admitted to hospital March 29, 1900. Family history good, both parents alive and well, and two brothers and three sisters are in good health.

He has never had any bad illness, nor suffered from anæmia previously. He has not had syphilis.

In 1898 he had a severe scalp wound which bled profusely, and about a year later he had profuse epistaxis from a blow from a stone.

For nine months he had been gradually losing strength, and had become anæmic. On exertion he had a sensation of tightness across the epigastrium, and pains in the back and legs. The feet and ankles became swollen towards night. For a week previous to admission he had been obliged to take to bed.

He was very pale in appearance; there was no jaundice. The pulse, 92, was soft and compressible, the tongue red and clean. Many of the teeth were carious.

The lungs were normal; systolic murmurs were audible at the base and apex of the heart, and over the great vessels. The lower edge of the liver could be felt, and the tip of the spleen came just below the costal margin.

The pulsations of the abdominal aorta were very plainly felt.

The urine was acid; Sp. gr. 1021; it contained a trace of albumen, no sugar, and showed a faint band of urobilin.

He was given iron and arsenic, and kept at rest in bed, and on this treatment he steadily improved. In June he left the hospital much better, but with still a trace of albumen in the urine, and tendency to œdema of the feet.

He returned and was re-admitted on April 25, 1901. He said that he had kept fairly well during the year, but had failed very much in the last six weeks. Since the beginning of the illness the slightest exertion had given [illegible] palpitation. Additional symptoms now were: twitching of the muscles of the legs at night, defective smell and taste, vomiting in the morning, a feeling of numbness in the fingers and toes, and loss of flesh. On examination he was well nourished; he was anæmic, but had not the lemon tint of pernicious anæmia; the pulse was 84, very compressible. The organs were in the same condition as previously. The tip of the spleen could just be felt. The knee-jerks and other reflexes were normal. The colon again contained scybala. The twitching of the leg muscles sometimes prevented sleep. The amount of urine passed daily was from twenty to forty ounces, it still contained a trace of albumin, and showed a moderately well-marked but diffuse urobilin band. Specific gravity 1025. There were no hæmorrhages in the fundus oculi, which was anæmic, but otherwise normal in both eyes.

From June 7 to 17 he was not so well, the temperature was raised to 100° with slight diurnal variations, and he was very weak, not being able to wash himself or get out of bed. There

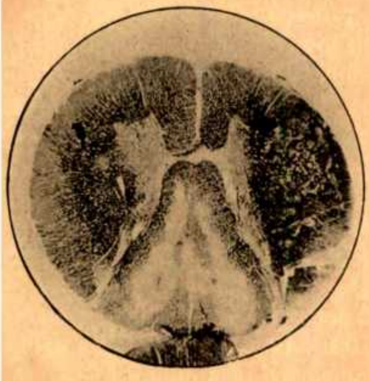

FIG. 1.

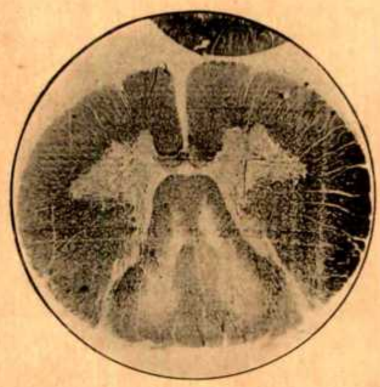

FIG. 2.

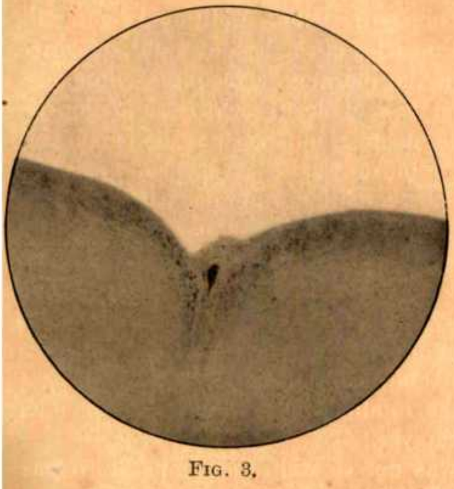

FIG. 3.

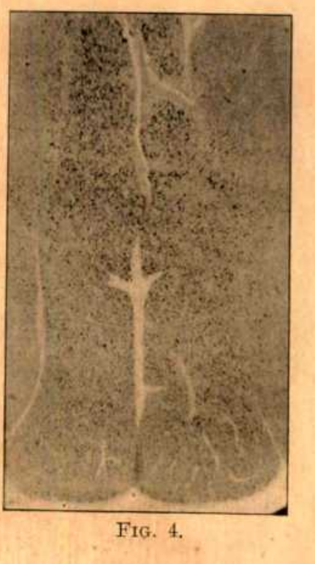

FIG. 4.

was also an increase of the urobilin in the urine, and the spleen became slightly larger.

From June 17 the condition of the blood gradually improved, he looked better, the spleen diminished in size, and the urobilin band practically disappeared from the urine. The skin was somewhat dark and pigmented. The muscles of the legs were weak and flabby, but not tender or distinctly wasted. Sensation was unaffected; the plantar reflexes were flexor in type, and feeble.

He now left the hospital, and was next admitted on April 3, 1902. He had kept in fair health until the beginning of the year, when he began to get very weak and to suffer from shortness of breath, palpitation, and giddiness.

He had a red flush on the cheeks, but the mucous membranes and the rest of the skin were pale. Over the chest, back, and limbs small round pigmented spots and petechiæ were fairly numerous. The pulse was regular, 76, full, and somewhat tense. The tongue was red and clean.

Physical examination showed that the condition of the organs was the same as on previous occasions, except that the liver was not felt. The spleen was slightly enlarged. The urine sometimes contained a faint trace of albumin. By Jaffé's test it appeared to contain a large quantity of indican, and with the spectroscope it showed a diffuse, but not intense or sharply-defined urobilin, band. There were also crystals of oxalates and phosphates.

During April and May his condition remained much the same; he was very weak and had no appetite. The temperature rose occasionally to 100° F. at night, falling a degree or so in the morning. He complained of numbness and tingling in the feet, especially in the right foot. Both optic discs and fundi were very pale, but there were no signs of past or present hæmorrhages. Towards the end of May he had a very bad attack of diarrhœa, after this he improved, and left the hospital at the end of June without any fresh symptoms. It should be added that at the beginning of June he was not so well, suffering much from pains in the back, abdomen, and limbs, and there was a rise of temperature to over 100° F. on several successive nights. The tingling and numbness in the legs and feet continued, but without objective loss of sensation, except that he had some difficulty in feeling a hot test-tube as hot. He was seen as an out-patient in October, and was then keeping well, though not very strong. His cheeks were red and his face generally a good colour; his chief complaint was of tingling and numbness in the feet and legs, and of some weakness. He attended as an out-patient for about two months. He could walk a fair distance, and there was nothing remarkable about his gait. The knee-jerks were present, not very active; there was no wasting of muscles and no rigidity. Plantar reflexes weak and flexor in type. Very slight defect of sensibility to touch and prick over the lower extremities, especially on the feet.

The condition of the blood at this date showed very marked improvement.

He was not seen again until May 14, 1903, when he was brought up to the hospital in an almost collapsed condition. He was very drowsy, and at first refused to take any nourishment.

There were signs of consolidation of the lower lobe of the left lung, and the breath and sputum were horribly offensive. The pulse was 120, very feeble, and the heart's sounds almost inaudible.

The temperature was irregular, soon after admission it rose to 103°, and for some days afterwards varied between 100° and 104°. He was very dusky in colour. The abdomen was retracted, the spleen was not felt. The urine was retained, a specimen withdrawn by catheter was acid, Sp. gr. 1,030, with much albumin and many hyaline casts.

The lung symptoms steadily developed, and he died from heart failure ten days after admission.

During this last stay in hospital he was too ill to make a satisfactory examination of the nervous system possible; he came in with a sacral bed-sore (he was very ill at home for three weeks before admission), and one developed on the right heel shortly afterwards. So far as could be made out there was no decided loss of sensation to touch or prick over the lower extremities, no spastic contracture of them, and he could move them well. There was general wasting.

The following table gives the condition of the blood at intervals during the whole course of the illness:—

Chart of Blood.

	Red blood corpuscles.	Hæmoglobin.	Blood decimal.	White cells.	
March 27, 1900	2,800,000	50 per cent.	1	10,000	Slight increase in eosinophiles, in lymphocytes and mononuclear cells.
June 11, 1900	3,000,000	55 per cent.	$\frac{11}{12}$	7,000	
May, 1901	2,240,000	32 per cent.	$\frac{13}{18}$	8,000	Lymphocytes and mononuclears increased somewhat. Eosinophiles in normal proportion.
June 3, 1901	2,000,000	38 per cent.	$\frac{19}{20}$		
June 20, 1901	1,700,000	34 per cent.	1		
July 15, 1901	3,800,000	52 per cent.	$\frac{13}{19}$	5,000	

	Red blood corpuscles.	Hæmoglobin.	Blood decimal.	White cells.	
May 23, 1902	2,000,000	30 per cent.	$\frac{3}{4}$	4,000	Differential count showed normal proportion between kinds of white cells.
Oct. 16, 1902	3,420,000	48 per cent.	$\frac{12}{17}$	4,000	
May 15, 1903	4,100,000	80 per cent.	1	20,000	Specific gravity 1,061. A polymorphonuclear leucocytosis due to pneumonic changes.
May 20, 1903	4,100,000	75 per cent.	—	20,000	

With regard to the red cells, at the beginning of the illness, and at each subsequent relapse, there were numerous microcytes, macrocytes, and poikiloytes, with a small number of normoblasts. As the condition improved on each occasion, the red cells tended to return to the normal shape and size. No megaloblasts were seen at any stage. The *post-mortem* appearances were briefly: gangrene of lower lobe of left lung; general bronchitis; congestion of base of right lung; heart muscle pale and flabby, but not fatty; congestion of liver and spleen; kidneys intensely congested and indurated; marrow in ribs, sternum, and shaft of one tibia red in colour.

Sections of the various organs were stained with hæmatoxylin and eosin, hæmatoxylin and rubin and rosin, and osmic acid and carmine.

The following is a short summary of the microscopical changes:—

Heart.—No fatty change; increase of pigment round nuclei of muscle fibres.

Liver.—Congestion; cloudy swelling of liver cells, many of which showed vacuolation.

Spleen.—Intense injection; some excess of large pale cells in sinuses, apparently due to proliferation of cells lining them.

Kidney.—Chronic glomerular nephritis of moderate extent, affecting the convoluted tubes but slightly.

Marrow.—Excess of polymorphonuclear cells.

No iron reaction given by any organ.

The brain was normal.

Spinal cord.—By the Pal-Weigert stain the posterior columns showed a degeneration which, beginning in the upper dorsal region (see fig. 7) as a small area of degeneration in the posterior and central parts of Goll's column, extended throughout the cervical portion of the cord. In the cervical region the degeneration was almost entirely one of Goll's column, except that it extended a little on the dorsal surface of the cord to involve the posterior end of the postero-external columns (fig. 6). The degenerated areas gave no reaction with Marchi's stain, most probably because the degeneration was of old standing, but in them were minute areas or cavities, from which the nerve fibres had disappeared. This same appearance was also noticed to a very small degree in the crossed pyramidal tracts for the extent of two or three segments in the upper dorsal region of cord only. The other parts of the cord were normal, as were also the gray matter, the anterior horn cells, and both anterior and posterior roots at all levels.

The interest of this case lies in the circumstance that the patient had recovered from his long continued anæmia, and died from an accidental lung affection. Further, the changes in the cord had not advanced very far, and apparently had ceased to extend; I think this is also the reason of the slight affection of the postero-external columns. The microscopic appearances and the clinical symptoms both point to the stationary character of the cord lesion. If this inference is correct, then the case illustrates the close connection of the cord changes with anæmia, and their dependence upon the latter. As showing the completeness of the recovery from anæmia the fact that the hæmato-poietic organs were able to cope with the strain of the fatal illness is important. Although in some respects, especially in the recurring episodes of blood destruction, the illness resembled pernicious anæmia, yet in most of its features, and notably in the fact of such complete recovery, it did not correspond with the latter condition. The state of the blood fairly corresponds to Cabot's third grade of secondary anæmia, the leucocytosis, which accompanies this form in the severer cases, not being very pronounced.

Case 3.—John C., aged 45, admitted under Dr. Harrison. The clinical notes of this case were very brief; it appears to have been entered in the hospital records as pernicious anæmia, but the notes, blood changes, and *post-mortem* appearances hardly bear out this diagnosis. The illness began nine months previously to admission with an attack of pneumonia, followed by severe anæmia, and for the last four months of this time he had had various paræsthesiæ, numbness and tingling in the hands and feet, with weakness of the limbs. There was no absolute paralysis, nor anæsthesia. He died of bronchitis, for which he came into the hospital, nine days after admission.

The blood examination gave 2,600,000 red cells; hæmoglobin 30 per cent.; blood decimal $\frac{3}{5}$. No increase of white cells. Red corpuscles varied in size; many poikilocytes; no megaloblasts or normoblasts.

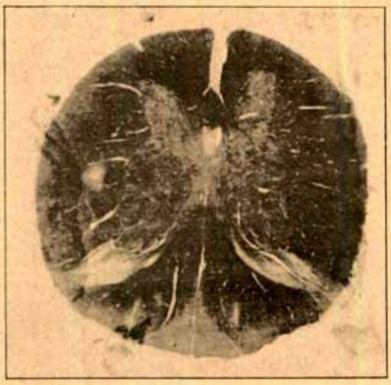

FIG. 5.

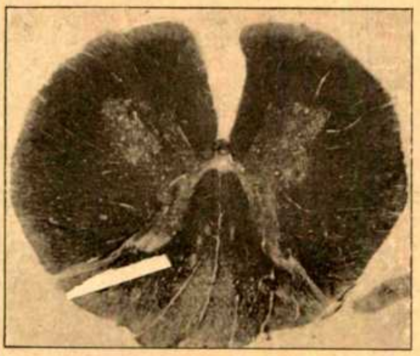

FIG. 6.

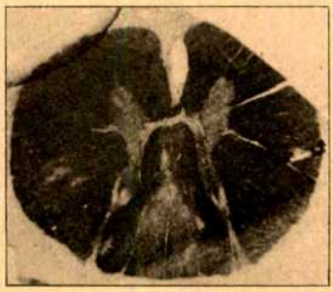

FIG. 7.

Post mortem.—The fat was bright yellow; some yellow transudate in serous cavities gave no urobilin band; general bronchitis; œdema of bases of lungs; no fatty changes present in the heart or other organs; kidneys very pale but not fatty; the liver and kidney gave a faint iron reaction, but not the spleen; the latter weighed 4 ozs., and appeared healthy; marrow healthy; small intestine showed patches of catarrh, no pink colour was obtained by treatment of mucous membrane with mercuric chloride solution; brain and cord healthy in appearance, except for the general anæmia, which affected every organ.

The above findings were confirmed by microscopical examination. The cord showed a more advanced degeneration of the posterior columns than in the two preceding cases, though here also the columns of Goll were most affected. The degeneration extended regularly, though slightly patchy in character, from the medulla to the level of the first lumbar roots, but not below this, being most intense in the cervical and upper dorsal regions. In the former the most intense degeneration occurred along the boundary between the postero-median and postero-external columns (fig. 8); it affected the greater part of these columns, leaving a marginal zone of varying extent, lining the posterior commissure and cornua and the dorsal surface of cord, free from disease. In the dorsal region the degeneration is rather less extensive, the marginal area being broader; the diseased part gradually diminished in passing down the cord until it became a very small patch on each side at the first lumbar segment, below which it could not be traced (figs. 10, 11).

In sections stained by Marchi's method a few scattered degenerated fibres were seen, as in case 1, in the lateral columns, (position of pyramidal tracts) and in the postero-external columns, in areas which by the Pal-Weigert stain appeared normal.

The degenerated fibres, as in the other cases, could be traced upwards to the posterior nuclei, chiefly nucleus gracilis, in the medulla, in which they ended (see figs. 3 & 4). The gray matter, posterior roots and other parts of the cord were healthy. For vessels, see general description.

Case 4.—This patient was one who died of anæmia in the hospital; but, unfortunately, I have no notes of the case, nor of the condition of the blood.

A complete examination was made of the spinal cord, brain, and some of the peripheral nerves. The brain and the nerves were healthy. In the cord degeneration of the posterior columns was found, extending through the cervical and dorsal regions, and through the lumbar as low as the level of the fourth lumbar roots, where it ended, not involving the sacral region. The degeneration in this case is more extensive than in the foregoing cases (see figs. 12, 13, 14), and corresponds in this respect more to case 5. It was, however, as in the milder cases, most extensive in the cervical region and upper dorsal regions, in which the whole of the posterior columns were affected, except a very narrow bounding rim of healthy tissue. In the lower dorsal and lumbar regions this margin is larger.

In the lateral columns in the upper and mid-dorsal regions of the cord there was a small area of degeneration on each side (not shown in the figures) in the position of the pyramidal tracts, but this did not extend into the other parts of the cord.

Case 5 is published in full in the *British Medical Journal*, vol. ii., p. 325, with figures of the spinal cord degenerations. In this case the condition was apparently one of pernicious anæmia. She died nine days after admission to the hospital. The duration of illness was 12 months, during the *last four months* of which she had suffered from increasing weakness of the legs, with a feeling of deadness in them; when admitted there was some loss of motor power, no affection of sensation, and the plantar and deep reflexes were active.

The blood examination on admission gave red corpuscles 870,000, many poikilocytes, macrocytes, microcytes, and some normoblasts. White corpuscles 10,000, the increase being due almost entirely to polymorphonuclear cells. Hæmoglobin 30 per cent.; blood decimal ·6. Seven days later red cells 830,000; whites 30,000; hæmoglobin 25 per cent. The changes in the cord were most advanced in this case, as a reference to the figures will show; briefly they consisted in a fairly complete degeneration, though somewhat patchy in distribution, of the posterior columns extending the whole length of the cord, ending above in the nuclei of the posterior columns in the medulla, and in the lower dorsal and upper lumbar region of a small area of degeneration in the posterior part of the left lateral column.

This series of cases, I think, shows that the spinal degeneration which occurs as a secondary change in profound anæmia, and has been found and described by many observers, begins in the posterior columns and first in the cervical and upper dorsal regions of the cord. Further, that in cases which die from some cause in an early stage of the cord disease, the affection may be unsuspected during life, and will be found limited to this region after death. In more advanced cases the posterior columns are involved almost throughout the cord, and the disease is then most extensive in the region in which it first begins, affecting here the whole of the posterior columns except a narrow rim of tissue on the dorsal surface and lining the posterior commissure and posterior horns; in the lumbar and sacral regions rarely spreading to so great an extent. These changes, though fairly regular, and possibly indicating that the fibres of the posterior columns which run the longest course are earliest degenerated, do not exactly map out special tracts, but are somewhat patchy in distribution, often varying at different levels for no obvious reason. The posterior roots do not seem to be affected. The appearances in some instances suggest some relation to the vessels, as the degeneration often seems to begin in the terminal distribution of the small artery that runs into the cord along the septum between the column of Goll and the postero-external column, that is to say, a small area about midway between the anterior and posterior ends of Goll's columns, involving slightly the postero-external columns. This relation is, however, though suggestive, not constant, and therefore no undue stress can be laid upon it.

In the cases in which the posterior column changes are most marked a patch of degeneration is also to be seen, either on one side or both, in the lateral columns in the position of the crossed pyramidal tract; this, again, is most evident in the cervico-dorsal or upper dorsal regions, but in one case appeared in the dorsi-lumbar region. It is more patchy and less regular even than the degeneration in the posterior columns, and does not extend over so great a length of the cord, in mild cases being limited to a few segments.[1]

It must be added to this that though the Pal-Weigert stain may indicate degeneration limited to the above situations, sections stained by the Marchi method often show a few scattered degenerated fibres occurring singly in a much wider distribution in the posterior, lateral, and occasionally a very few in the anterior, columns. Fibres stained black by this method also appear, if the case is a recent one, in the areas which by the Pal-Weigert stain appear to be already degenerated (see fig. 4). As regards the finer changes they may be said briefly to be practically identical with those described by Dr. Risien Russell in cases of "subacute combined degeneration" of the cord. The vacuolation resulting from the destruction and dropping out of nerve fibres is generally well seen.

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[1] A typical example of the affection of the pyramidal tract in these cases is figured in my paper in *Brit. Med. Journ.*, 1897. Vol. ii., p. 326, fig. 3.

In the degenerated areas there is an increase of the neuroglial framework, the vessels often show greatly thickened walls, and sometimes hyaline degeneration of them. In some of my sections the overgrowth of neuroglia seems to have been more extensive than was met with by Drs. Putnam and Taylor.[1] The older the patch the greater seems to be the sclerosis, the denser the structure of the part, and the less marked the vacuolated spaces, as if the tissues had contracted. In such cases the increased connective tissue may appear to spread directly from the vessel walls, and sometimes a leucocytal infiltration is seen around the latter. These changes seem to me to be entirely secondary to the parenchymatous degeneration of nerve fibres, and although, as alluded to above, this degeneration shows an apparent relation to vascular distribution, I should not attach further weight to this than that it may possibly indicate the route of conveyance of a toxin.

In favour of the secondary nature of the vascular changes is the fact that they are not evident in regions where the nerve degeneration is slight (and recent), and, further, are not present in areas of the cord where the only evidence of disease lies in the presence of a few scattered degenerated fibres. I found no patches of myelitis in my cases. The gray matter and its contained cells showed no important changes, the only noticeable one being the not infrequent presence in the cells of the anterior horn and of Clarke's

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[1] "Diffuse Degeneration of the Spinal Cord." *J. Nerv. and Menta Dis.*, 1901.

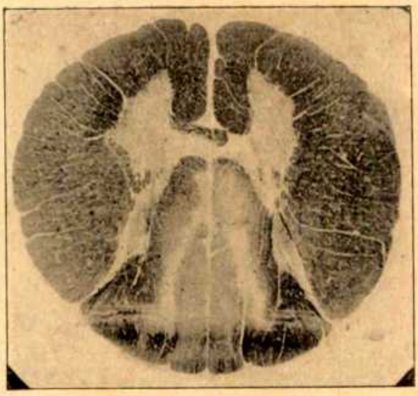

FIG. 8.

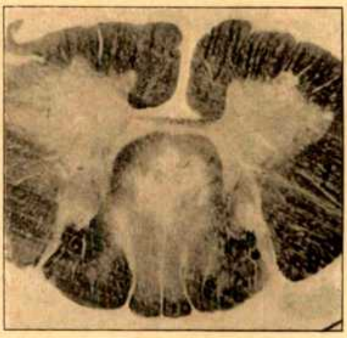

FIG. 9.

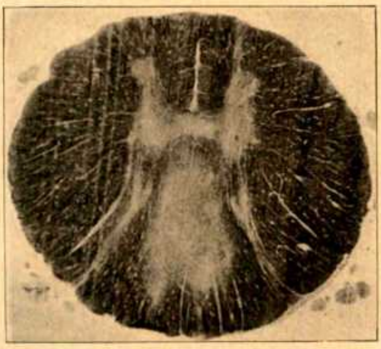

FIG. 10.

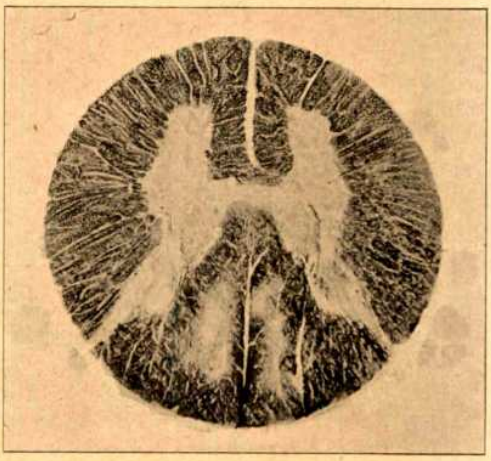

FIG. 11.

column of an unusual amount of pigment. This pigmentation is also mentioned by Putnam and Taylor. The meninges were in all cases unaffected, and the peripheral nerves, where examined, normal.

To turn to the clinical features of these cases, clinical symptoms may certainly be absent, and the lesions in the cord not even visible at the *post mortem*, but only found after hardening and staining it. It is probable, therefore, that such degenerations in the cord are of more frequent occurrence in patients dying from anæmia than is generally supposed. As these changes are secondary and subsidiary to the general symptoms of severe anæmia, they may easily be overlooked, but my more recent experience is that a careful investigation will generally elicit some signs of the affection of the cord.

The most prominent clinical symptoms are certainly paræsthesiæ of various kinds, such as numbness, tingling, feelings of pricking, or of pins and needles, first appearing in the feet and legs. Later the hands and forearms may be affected, when a common complaint is, that numbness or deadness of the fingers renders fine movements difficult. These paræsthesiæ vary much in intensity during the course of the illness. Objective disturbance of sensation is not marked, but in the late stages may consist of some deficient appreciation of all forms of sensation over the lower extremities. After these sensory symptoms weakness in the legs may follow, often in the sense that they feel heavy, and later still both arms and legs may be weak. A frequent and often troublesome symptom, is twitching of the muscles, especially of the legs, and at night. The deep reflexes are present, ankle clonus is not obtained, and the plantar reflex, flexor in type, is sometimes difficult to get. The knee-jerk was absent towards the end of life in case 2, but he could only be examined as he lay in bed.

There is no ataxy, and no incontinence of urine.

In relation to the clinical symptoms, I should like to briefly mention another case, th iagnosis was not confirmed by a pathological e

The patient was a middle suffered from a form of rhinitis, with a spongy condition of the nasal mucous membrane, which gave rise to recurrent attacks of profuse epistaxis. Under treatment in Hospital he was invariably relieved, but after an interval, generally of considerable duration, six months to two years, he relapsed, and the attacks of epistaxis returned. This epistaxis led to moderately severe anæmia of the chlorotic type, as regards the ratio of red blood cells to hæmoglobin. He was thus under observation for anæmia at intervals for over five years. The following is the chart of the blood, which indicates a progressive deterioration of it :—

	Red Cells.	Hæmoglobin.	White Cells.	
1896. Jan.	3,100,000	31 per cent.		
(On leaving Hospital) March	4,800,000	62 per cent.		
1899. Feb.	2,400,000	25 per cent.	10,250	Many poikilocytes, normoblasts, small and large red corpuscles.
			Polymorphonuclears 69 per cent. Small lymphocytes 23 per cent. Large lymphocytes 4 per cent. Eosinophiles 4 per cent.	Poikilocytes, normoblasts, micro- and macrocytes numerous.
1900. July	2,750,000	35 per cent.	9,000 Differential count normal.	As before.
1901. Feb.	2,400,000	25 per cent.	12,000	
			Polymorphonuclears 67 per cent. Small lymphocytes 23 per cent. Large lymphocytes 8 per cent. Eosinophiles 2 per cent.	As before.

On the last occasion the anæmia only slightly improved under treatment, but on previous ones recovery was satisfactory, as in 1896, but I have not thought it necessary to give the actual blood counts each time.

Whilst in the Hospital in July, 1900, he complained for the first time of various paræsthesiæ, especially tingling and numbness in t[redacted]d legs; and on his subsequent admission in 1 toms had very much increased, and were the t discomfort. He now complained of deadness or numbness of the hands and fingers, varying from time to time, and sometimes interfering with the performance of fine movements. There was no decided objective loss of sensation, although it was perhaps somewhat dull over the feet. He also complained of some weakness in the legs, so that he could not walk so far as formerly. There was no ataxy and no muscular wasting; the knee-jerks were slightly exaggerated.

I have not been able to trace this man, but I have no doubt that the case was one of the same kind, *i.e.*, of a spinal lesion developing secondarily to repeated attacks of severe anæmia.

Hæmorrhage as the cause of anæmia in such cases deserves special mention, and is probably of importance; it occurred in two of my cases as the prominent etiological factor, and has been noticed in several others.[1]

The cases that I have described above form a fairly distinct group, and the changes in the cord occur in such a way as to show that they result from anæmia, and from forms of anæmia due to blood destruction. Though pernicious anæmia is one form which causes these cord lesions, it is by no means the only one. I am here taking pernicious anæmia in the ordinary sense of the term, though there is so much difference of opinion as to what actually constitutes pernicious anæmia that it is difficult to avoid confusion. In favour of the point that it is only when anæmia is due to blood destruction that these cord changes follow is the striking fact that they are not seen in chlorosis, of however long standing. A possible view of the pathology of these cases seems to me to be that of parenchymatous degeneration due to some toxin, possibly set free in the course of a pathological blood destruction, and therefore hæmolytic in origin. More extended observations on the blood, and on the precise character of the anæmia, would tend to elucidate the nature of the process.

In this particular group the cord changes often follow on so long a period of anæmia that it is difficult to suppose that the lesion is other than a secondary one, although it is possible that the cord changes might be due to the continued action of the same cause that has already produced anæmia, just as in those cases of cord degeneration and anæmia described by Dr. R. Russell and Dr. J. Taylor, they suppose that both conditions are due to the same toxin.

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[1] *Vide* Dr. James Taylor, "A Case of Subacute Combined Sclerosis." BRAIN, 1904, p. 84.

It may be mentioned, in passing, that in pernicious anæmia lesions in the spinal cord of quite another kind sometimes occur, namely, numerous small hæmorrhages, chiefly in the gray matter. Such cases are rare[1]; the hæmorrhages are to be compared with those found in the retina in this disease, and seem to me to have nothing to do with the cases now under consideration. There was no evidence in my sections that the degeneration started from any lesions of this kind

The question now arises: Is there sufficient reason to put cases such as are considered in this paper, in which the cord changes distinctly follow or are secondary to profound anæmia of some duration, into a separate group, and to distinguish them from those in which similar lesions of the spinal cord occur, apart from anæmia; and further, what is their relation to the cases described by Risien Russell, Batten, and Collier[2] under the title of "Subacute Combined Sclerosis," and by Putnam and Taylor[3] and others, under the head of "Diffuse Degeneration of the Spinal Cord?" The latter writers say that "it seems to us too soon to make either clinical or pathological divisions in the general group of diffuse degenerations, and particularly to attempt the demonstrations of lesions peculiar to anæmia."

Such a division has been already attempted by Bastianelli in 1895, but his distinction, valuable as it is, is partly based on the acute or chronic course of the disease,[4] and will hardly hold good, for, as three of my cases show, and as other writers have also pointed out, the anæmic cases which, according to his classification, fall into the

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[1] For a case of this sort see *British Medical Journal*, 1897, vol. ii., p. 326.

[2] BRAIN, 1890, Spring.

[3] *Journ. Nerv. and Ment. Dis.*, 1901.

[4] See J. Taylor, "A Case of Subacute Combined Sclerosis," BRAIN, Spring, 1904.

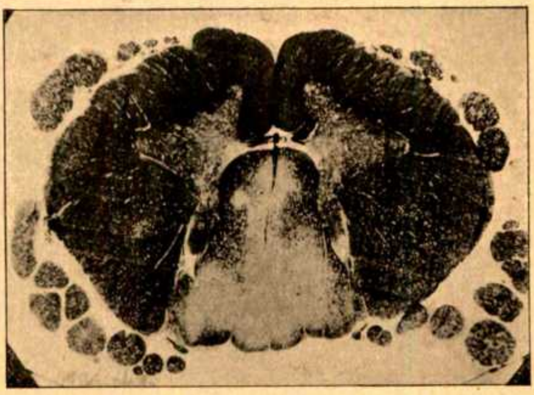


FIG. 12.

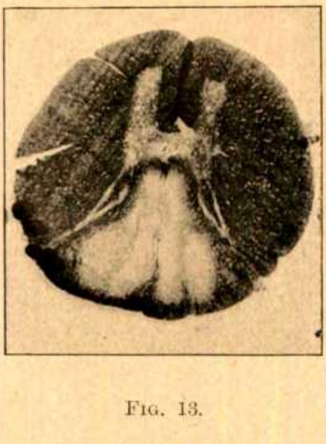


FIG. 13.

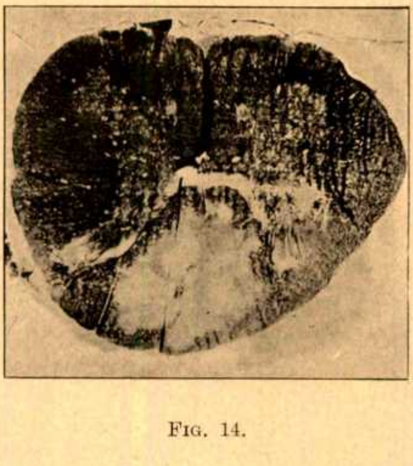


FIG. 14.

acute class, are, on the contrary, often chronic in course. I believe, however, he is correct in the view that, as a general rule, in the cases secondary to anæmia the cord changes are slighter, and most often limited to the posterior columns.

Without laying undue stress on the anatomical characters of these cases, it seems to me that there are good grounds, both clinical and pathological, for separating them from similar forms of degeneration of the cord, and that these are:—(1) The presence of decided or severe anæmia as the first, or sometimes the only, sign of illness; preceding any evidence of spinal cord lesion. (2) Clinically symptoms and signs of disease of the cord are never very pronounced, and may be absent, though, possibly, in most cases are present when looked for. (3) Anatomically the lesion is generally confined to the posterior columns, and, if it affects the lateral columns, only does so to a small extent. And (4) it differs in extent and distribution from *subacute combined sclerosis* or *diffuse degeneration of the cord* in the following ways, according as the disease in the latter case is slight or severe. (*a*) In the milder cases of diffuse degeneration the degenerations are more precisely limited to the neuronic systems of the cords; thus, in two cases under my own observation the columns of Goll were diseased from the lumbar region upwards to the medulla, and the crossed pyramidal and dorsal cerebellar tracts were similarly affected; whilst in (*b*) the more severe cases, the degeneration is both much more extensive than in the cases considered in this paper, and also affects the periphery of the cord most, and appears to spread thence inwards, at any rate in the lateral and anterior columns.

On the other hand, it must be admitted that the microscopical details of the degeneration process appear to be similar in both groups, and further that there are cases which occupy an intermediate position, in which the cord changes are associated with profound anæmia, and are also very extensive in distribution. It seems to me that the chief difficulty lies in the nature of these intermediate cases.

In conclusion, although the evidence is not sufficient for a final classification, I think that a step in advance

ILLUSTRATIONS.

Photo-micrographs of Cord.

CASE 1.

Fig. 1.—Cord at level of third cervical.

Fig. 2.—Cord at level of fourth and fifth cervical.

These figures show the point of commencement of degeneration as described in the text.

CASE 2.

Figs. 5, 6, 7.—Sections of cord at level of second cervical, third and fourth cervical, second dorsal.

(In section of second dorsal there is a light streak across dorsal free edge of left lateral and posterior columns due to irregularity in thickness of section.)

CASE 3.

Sections of cord at level of:—*Fig.* 8, third cervical. *Fig.* 9, seventh cervical. *Fig.* 10, seventh dorsal. *Fig.* 11, first lumbar: This photograph is too light in the anterior and lateral columns which were perfectly healthy.

Fig. 3.—Marchi stain. To show degenerated fibres ending in nucleus gracilis.

Fig. 4.—Marchi stain. To show about the number of fibres which stain black by Marchi in a degenerated column of Goll, which is also shown stained by Pal-Weigert's method in *Fig.* 8.

CASE 4.

Fig. 12.—Cord at level of eighth cervical.

Fig. 13.—Cord at level of tenth dorsal.

Fig. 14.—Cord at level of second lumbar.

A similar degeneration extended through the cervical region to the medulla.

SYSTEM LESIONS OF THE POSTERIOR COLUMNS IN GENERAL PARALYSIS, AND THEIR BEARING ON THE POINT OF ORIGIN OF TABES DORSALIS.

BY DAVID ORR, M.D.
AND
R. G. ROWS, M.D.

For the last three years, and more especially during the last year, while investigating the degenerations of the posterior columns in general paralytics, we have examined several cases in which the lesion was sufficiently pronounced to allow us to follow the course of the diseased fibres, and to justify us in coming to certain conclusions with regard to the origin of the earliest changes affecting the sensory spinal neurons of the first order.

We have found that the degeneration of these neurons has always begun at one definite spot in every case we have examined and in every segment of the various cords.

The reason why the starting point of these degenerations should be so constant will be pointed out in the course of the paper, and certain reasons will be advanced to show why the fibres should be vulnerable at this point. All the facts which we shall bring forward have been ascertained by an accurate examination of serial sections. Each cord has been examined from the lowest to the highest segment.

As we soon realised that no sclerosis was present in the majority of our cases, *i.e.*, that the lesions were in the early degenerative stage, all our tissues were submitted to treatment by the Marchi method, but care was taken to supplement this reaction with the Wolters and Van Gieson methods. The posterior roots were treated in exactly a similar manner.

The amount of lesion which we have found has naturally varied, a fact which has been of the greatest assistance in the study of its exact starting point and in the localisation of the degenerated fibres in their first course in the cord.

Our results have been so constant, and the areas occupied by the degenerated fibres have so absolutely coincided with the sclerotic areas found in early cases of tabes dorsalis, that we have thought it worth while to record our results and conclusions.

We gave a preliminary account of these acute lesions at the meeting of the British Medical Association, held in Oxford last year (1), and since then we have completed the study of cases, in some of which sclerosis was beginning to replace the degeneration; in others the sclerosis was well established. We have, therefore, been able to follow the process in all its phases, from the slightest degree of degeneration onwards.

A study of the literature of tabes dorsalis shows clearly that there is considerable diversity of opinion with regard to the starting point of the process, but on one point all authorities are agreed, viz.: that the lesion is systemic and affects primarily the sensory fibres of exogenous origin. This fact at once paves the way for further research, because a lesion of this system is easily followed, on account of the accurate anatomical work which has been done since the introduction of the Golgi and Marchi methods. Bearing these two facts in mind, we can say that any lesion of this system which is of sufficient degree to enable us to map out its limits in the posterior columns, and to follow the degeneration of the terminals and collaterals of the fibres into the gray matter where they arborise around the nerve cells, is indistinguishable from a tabetic lesion in its course and distribution. It is of importance, therefore, that these early lesions should receive special attention, because their starting point is easily defined. On the other hand, we consider that the continued study of cases of tabes, in which sclerosis has replaced the degenerative phase, is of little assistance in enabling us to determine the pathogenesis of this disease.

Every one must admit that by the time a pathological process has reached the stage of sclerosis many secondary factors have been introduced—which re-enforce the original exciting cause—and that an extension of the pathological process must have taken place in many directions.

The only accurate means by which we can determine the starting point of any disease, whether in the nervous system or elsewhere, is to first study it in the early degenerative phase. It is a neglect of this which accounts for the many theories which have been advanced regarding the pathology of tabes dorsalis.

Of the cases of general paralysis which we have examined it is only necessary to give one in detail, as the degenerations in the others affected the same system, and only differed in degree. The results obtained in this case by the Marchi method, and which may be seen in the accompanying photographs, were as follows:—

In all the sacral segments, the areas occupied by the sensory fibres of exogenous origin are thickly dotted with black droplets; scattered between these are many normal fibres.

At this level the exogenous fibres derived from the internal division of the posterior root are seen curving round the inner margin of the substantia gelatinosa Rolandi, and running forwards to occupy almost the whole breadth of the columns. They do not quite reach the commissure anteriorly nor the posterior periphery of the cord, and along both sides of the median septum a narrow strip is left practically free from degeneration. With the exception of these three areas, which constitute the endogenous systems, the whole column is well filled with degenerated fibres.

In the endogenous areas—whose limits will be shown later—there are very few degenerated fibres, so that these zones are sharply defined, and their limits are easily traced. The degeneration of the fibres of the exogenous system is seen to commence just at their point of entrance into the cord, and, in the extra-medullary part of the root, no degeneration is present except in a small segment close to the cord.

Springing from the degenerated areas in the posterior columns, many fibres can be seen running into the gray matter. These first become evident in the fifth sacral segment, and proceed principally from the antero-lateral angle of the column, but also to a less extent from an area just anterior to Rolando's substance. In the first sacral segment these degenerated collaterals are seen in greater numbers. A few traverse the internal corner of the substantia gelatinosa, but the great majority curve out into the posterior horn just anterior to this structure, and pass forwards to all regions of the anterior cornua. In the fourth sacral segment these collaterals could only be traced as far as the lateral group of cells in the anterior horns and, in the fifth sacral, the degeneration was confined to the posterior horn.

In Lissauer's area not a single fibre shows the Marchi reaction, and by the integrity of this region the rows of altered myelin in the internal division of the sensory root are thrown into bold relief. It might be stated here that not only in the sacral but in all other regions of the cord, Lissauer's tract shows practically no degeneration.

As mentioned above, the degenerated exogenous sensory system is sharply marked off from the endogenous. The anterior endogenous systems in the second sacral segment occupy the area just behind the posterior commissure, and extend for a short distance along both sides of the median septum, while the posterior systems occupy the postero-mesial parts of the columns and an oval area just anterior to this.

In the lumbar region the Marchi reaction of the lumbar root fibres is found scattered over the same areas as in the sacral segments; the degenerated exogenous fibres of the lower roots now occupy the regions by the median septum. Both the anterior and posterior endogenous systems pass lateralwards. Further, the degenerated fibres passing into the gray matter are more numerous, and can be seen streaming through the inner margin of Rolando's substance, and from a point just anterior to this. At the level of the second lumbar segment the degeneration reaches as far inwards as the median septum and as far forwards, in the middle line, as the posterior commissure. An area just behind the outer part of this commissure and extending for a short distance backwards immediately internal to the base of the posterior horn is found to be entirely free from degeneration with the exception of a few isolated fibres. This is the cornu-commissural layer of endogenous fibres now occupying a more lateral position than in the sacral region.

Just behind the endogenous tract at this level of the cord, a well-marked bundle of degenerated fibres makes its appearance. This arises from the cuneate fasciculus and passes into the gray matter to arborise around Clarke's nucleus. The terminal arborisations of these fibres amongst the cells of the nucleus are well shown by the Marchi method. With the displacement of the posterior endogenous system outwards, the degenerated exogenous system extends further inwards and more towards the periphery of the cord, so that the area formerly occupied by Flechsig's oval is now filled with degenerated fibres.

In the first lumbar segment the degeneration in and around Clarke's nucleus, and of the fibres passing forwards to terminate around the cells in the anterior cornua is especially well marked.

From this point upwards there is a steady decrease in the degeneration affecting the intramedullary part of the roots and middle root entry zones, with a consequent diminution in the number of degenerated fibres passing into the gray matter.

At the level of the tenth dorsal segment the degeneration in the root entry zone is distinctly less, but more marked on the left side, and does not extend so far internally and posteriorly as in the lumbar region. A triangular area around the hinder portion of the median septum is thus left whose base is formed by the periphery of the cord, and in which the preponderance of normal fibres is much greater than in the entry zones, fig. 4. The separation of the degeneration in the most internal part of the posterior columns from that in the entry zones is more distinctly seen a little further up the cord, and in the sixth dorsal segment we find a very well defined area almost entirely free from Marchi reaction between these two regions.

In the anterior part of Goll's column the degenerated fibres are collected around the median septum, but in the posterior part they are scattered somewhat diffusely.

In the second dorsal segment there is still less degeneration in the entry zone which is more evident, however, on the left side. In Clarke's column and in the anterior cornua, the degeneration is very slight. The anterior and internal parts of the cuneate fasciculus—just behind the posterior commissure—contain a very considerable number of degenerated fibres, while in Goll's column the large majority of the degenerated fibres lie along the posterior third of the median septum and spread outwards from it for a little distance at the periphery of the cord. In the first dorsal segment the degeneration in the posterior columns, with the exception of that in Goll's tract, is again reduced. In Clarke's column of cells there is only a trace of Marchi reaction.

On passing from this segment up into the eighth cervical we find a return of degeneration in the entry zone and in the intramedullary part of the root. This can be traced back as far as the point of entrance of the fibres into the cord, but not beyond. The degeneration is more marked on the left side, and many degenerated fibres can be traced into the posterior and anterior horns.

In the seventh cervical segment the degeneration is seen to pass inwards, and the degenerated fibres are found scattered in the anterior part of the cuneate fasciculus on both sides as far forwards as the posterior commissure; and along the inner margin of the cuneate fasciculus where it lies adjacent to the column of Goll the fibres form a distinct band which, however, is more pronounced on the left side. Degeneration in the gray matter is still present. There is a slight increase of degeneration in the right root at this level.

In the sixth cervical segment the degeneration in both entry zones is diminished, and the band of degeneration along the inner margin of the cuneate fasciculus is not quite so well defined on the right side. On the left side there is a very distinct layer with its broad end directed towards the commissure and its tail pointing backwards. In Goll's column the degenerated fibres form an L-shaped figure on both sides of the postero-median septum. The degeneration in the gray matter is much diminished. A little further up the cord the degeneration in the entry zones disappears, and the layer along the inner margin of the cuneate fasciculus diminishes, while in the anterior part of Goll's tract at the level of the second cervical segment there is a sensible increase in the number of degenerated fibres.

In the posterior roots—outside the cord—treated by Marchi's, van Gieson's, and Wolters' methods, there is not sufficient change to account for the intramedullary lesion. Marchi's method shows a few scattered droplets while the two latter methods yield almost entirely negative results in both cord and roots.

To briefly recapitulate the points of importance in the case, we find:—

(1) Degeneration of the internal division of the sensory roots in their intramedullary path, commencing at the point of entrance into the cord. The maximum of degeneration in this system is found in the lumbar region, and it becomes progressively less in the dorsal cord from below upwards. At the eighth cervical segment there again occurs a slight increase, as shown by the Marchi reaction in the intramedullary part of the root, and by the degenerated layer along the inner margin of the cuneate fasciculus in the higher cervical segments.

(2) Complete integrity of the external division of the sensory root which consists of fibres of small calibre, and, in the cord, forms Lissauer's tract.

(3) The collaterals and terminals of the sensory system which pass into the gray matter, share in the degenerative process, viz.,

those passing into the posterior and anterior horns, and those arborising around Clarke's nucleus.

(4) The long fibres derived from segments in the lower parts of the cord are affected, as shown by the position of the degeneration in Goll's tract in the cervical region. This degeneration is small in amount, owing to the fact that under normal circumstances only a few fibres of the lower roots ever reach the higher segments of the cord, the great majority terminating around cells in the gray matter at the various cord levels, and also because not all the fibres in the internal division of the roots of this case were attacked.

Taking into consideration the integrity of Lissauer's area and the absence of degeneration in the extramedullary part of the roots, it is obvious, therefore, that the internal division of the sensory fibres has been attacked at its point of entrance into the cord, and that in consequence of this injury an upward degeneration has taken place, reaching as far as the terminals and end arborisations.

In all the cases besides the above, examined up to the present time, we have noticed a striking similarity with regard to the point at which the degeneration of the intramedullary portion of the posterior root fibres has begun, and in the distribution of these fibres in their course up the cord. Whenever a degeneration has been found it has always begun at the point where the fibres have entered the cord, and has been traced along a definite course.

We shall be better able to understand the distribution of the lesions if we briefly consider the path of the peripheral sensory neurons in the cord.

The posterior columns contain two sets of fibres, and each of these has a distinct origin and a definite course in the cord. One set is derived from the cells of the posterior root ganglia, and reaches the cord through the posterior roots. These are the exogenous fibres. The other takes its origin from the cells of the gray matter of the cord, and is, therefore, described as endogenous.

The septo-cornu-commissural endogenous system occupies a different position in the various regions of the cord. In the lower sacral region it is septo-commissural, in the lumbar region it is cornu-commissural, and in the higher portions of the cord it spreads further back along the inner margin of the posterior horn.

The posterior endogenous system in the lower part of the cord is represented by the oval field of Flechsig and the triangle of Gombault and Philippe.

The embryological researches of Flechsig and Trepinski have led them to describe four zones in the posterior columns, which are determined by the period at which the fibres receive their myelin sheath. These zones, however, contain both exogenous and endogenous fibres, and are known as the posterior root zone, which consists of a lateral and a median portion—the anterior root zone, the middle root zone, and the median root zone.

Homén (2) and Philippe (3), in their articles on tabes, state that this division of Flechsig is not sufficient to indicate the course of the exogenous fibres in the posterior columns which are affected in this disease, and they suggest that it is necessary to follow the anatomical distribution of the exogenous system.

The first zone which the root fibres occupy after they have entered the cord is spoken of by Homén as the "root-entry zone" "Würzeleintrittszone" (fig. 1A). This root-entry zone lies along the inner and hinder portion of the posterior cornu. Medialwards it extends, more or less, into the column of Burdach. In the lower segments of the cord it is somewhat oval in shape, but in the upper parts it is narrower and elongated sagitally.

As the fibres ascend the cord they pass inwards and forwards along the inner margin of the posterior cornu, being pushed in these directions by the fibres of the succeeding roots.

From this zone many fibres, terminals and collaterals, stream into the gray matter of the posterior horns. The fibres which enter from the posterior part of the root-entry zone pass through the tip, and those from the anterior part through the inner margin of the cornu.

Some of these fibres arborise around the cells of the gray matter of the posterior horn; others pa[illegible]rds to the cells of the intermedio-lateral regio[illegible]rior cornu, and many go to Clarke's nucleus.

The longest fibres of the posterior roots then form a long, narrow band, curved with its concavity outwards, which extends from the cornu-commissural angle in the anterior root zone, backwards through Burdach's column to the periphery of the cord. The fibres of this band continue to be pushed inwards and backwards by the successive roots which enter the cord, and the longest fibres of the posterior roots finally occupy the positions indicated in the accompanying diagram (fig. 1A).

Now, when we turn to the sections of the various cords which we have examined, we find that the areas occupied by the degenerated fibres correspond exactly with those which have been mentioned above in describing the intramedullary path of the posterior sensory neurons. Moreover, we are able to trace the course of the short and long fibres, terminals and collaterals in these areas by the Marchi reaction.

The degeneration of the fibres in these cases has invariably begun at the point where they dip into the cord.

Thus, in the case detailed, in the sacral and lower lumbar segments much degeneration is present in the intramedullary portion of the posterior root fibres, the root-entry zone of Homén.

We would state here that by the term root-entry zone we wish to indicate the area outlined in the diagram (fig. 1A).

In the second sacral segment (fig. 2) it will be seen that there are numerous affected fibres in the root-entry zone, around the angle of the posterior horn and along its inner margin. But the area behind the posterior commissure and inside the neck of the posterior horn on both sides, together with the areas on each side of the median septum and along the periphery of the cord, contain very few degenerated fibres. These are the endogenous areas of the second sacral segment.

In the second lumbar segment (fig. 3) the root-entry zones are still thickly studded with degenerated fibres, as also are the areas along the anterior half of the median septu[illegible]se are the long fibres of the roots which have ent[illegible]n and have been pushed inwards.

The anterior endogenous system is here cornu-commissural in position, *i.e.*, it has passed outwards, and now occupies the area inside the cornu-commissural angle and the inner margin of the posterior horn for some distance backwards. This area is easily distinguished by the absence of degenerated fibres.

The posterior zone, again, contains only a few scattered droplets.

As we pass further up the cord the posterior columns contain fewer degenerated fibres. This is due to the fact that there is less degeneration in the intramedullary roots, and also that fibres are continually passing out of the columns into the gray matter.

It is obvious, therefore, that the degenerated fibres situated in the parts internal and posterior to the entry zone are fibres of longer course, which have entered by lower roots, and some of which will reach the highest levels of the cord in Goll's tract.

But although there is a progressive diminution in the amount of degeneration as we ascend the dorsal region, we find that in certain roots there is a definite increase, which gives rise to a corresponding increase of degeneration in the root-entry zone. This allows us to follow more clearly the course of the fibres in the first few segments after they have entered the cord. For example, the left eleventh dorsal root contained more affected fibres than the tenth or ninth, and the fibres of this root can be traced inwards and forwards along the inner margin of the posterior horn in the next two or three segments.

Again, the intramedullary portion of the right eighth dorsal root fibres (fig. 4) also showed more degeneration than the adjacent roots. These fibres could be followed inwards and forwards as they ascended the cord, and at the sixth dorsal they reached to the commissural angle.

These variations in the amount of degeneration of the intramedullary portion of the sensory neurons are important, and explain the curious changes in the distribution and amount of degeneration which may be present in any segment of the cord, and which sometimes makes it difficult to understand the systemic character of the lesion.

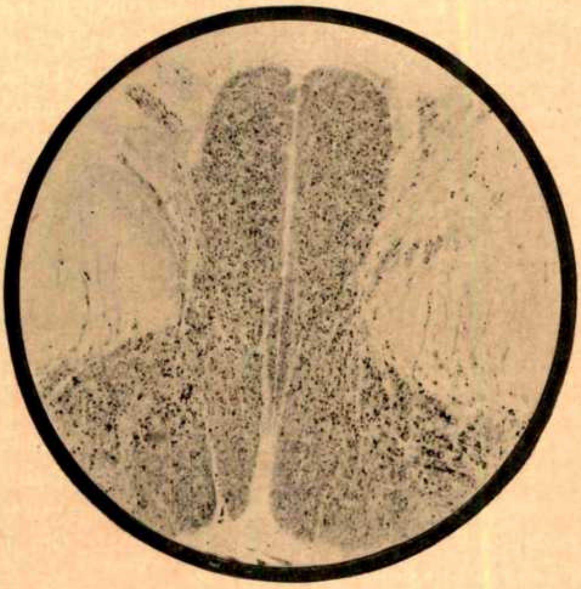


FIG. 2.
Second sacral. *Photomicrograph.*

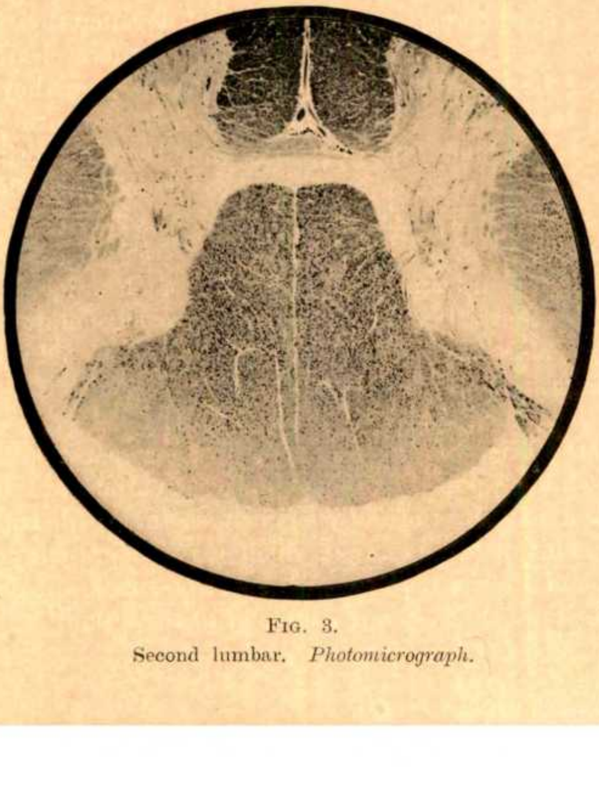


FIG. 3.
Second lumbar. *Photomicrograph.*

It will be remembered that on reaching the upper limit of the dorsal region the lesions in the posterior columns were less marked than in any other portion of the cord.

Immediately we examine the lower cervical region we find that there is again an increase of degeneration in the intramedullary portion of the root fibres, and in the root-entry zones. This gradually passes inwards and forwards, and ultimately forms a definite band along the inner margin of Burdach's column (fig. 1B).

In Goll's column the degenerated fibres, which are derived from the lumbar and sacral roots, lie in its internal and posterior portion.

Between this zone and the degeneration in the internal part of Burdach's column, there is an area in which very few altered fibres are found. This is the area normally occupied by the dorsal roots 12-5 [Edinger (4),] (fig. 1A & B). The absence of degenerated fibres in this area is explained by the fact that the intramedullary portion of the fibres of the dorsal roots were much less affected than those below.

We must now turn again to the various segments to consider the course of the terminals and collaterals which enter the gray matter, as they are demonstrated by the Marchi reaction in our cases.

In the second lumbar segment (fig. 5) numerous degenerated fibres, terminals and collaterals, may be seen passing from the root-entry zone, some through the substance of Rolando, and forwards through the gray matter; others through the inner border of the posterior horn, and then forwards for various distances.

This picture is not confined to any particular segment, but is invariably seen in every segment in which there is much degeneration present in the root-entry zone.

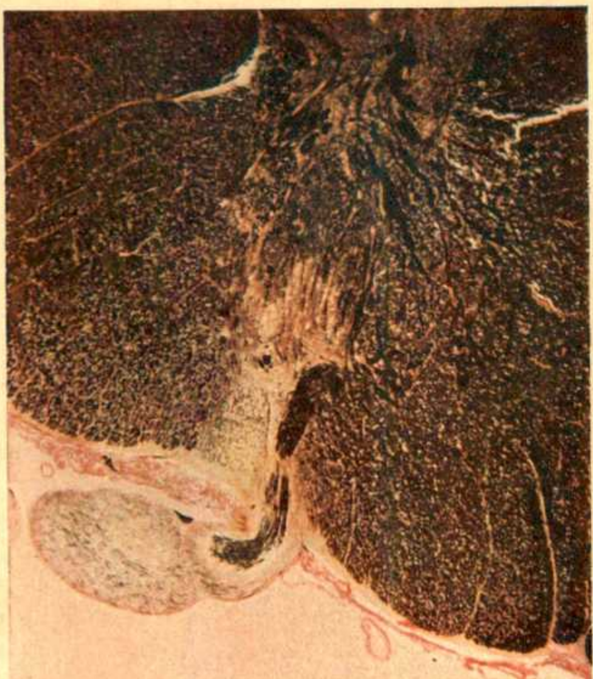
By referring to the photographs which are published with this paper, it will be possible to follow the course of these degenerated fibres in the gray matter. Most of those which pass into and through the substance of Rolando end by arborising amongst the cells of the posterior horn.

Of those which enter through the inner margin of the posterior horn, some end in the adjacent gray matter, but many have a longer course.

The fibres which enter at the angle of the posterior horn pass outwards and forwards towards the intermedio-lateral region; others forwards to the anterior cornua. But the most noticeable are the numerous affected fibres which pass into Clarke's nucleus. These are well seen in fig. 6, which is a photograph of Clarke's column in the first lumbar segment.

We have now followed, by Marchi's method, the course of the intramedullary portion of the exogenous fibres from the point at which they have entered the cord to their termination, and we find that the course of the fibres in the posterior columns, and of the terminals and collaterals in the gray matter, corresponds absolutely with the paths of these fibres as they have been described in the most recent anatomical works (Ramon y Cajal, Golgi, Edinger, Lenhossék, van Gehuchten, Homén, and others). We have pointed out also the areas which the endogenous fibres occupy in the posterior columns, and we have shown that these areas contain few degenerated fibres in all our cases. Moreover, the tract of small fibres known as Lissauer's tract, which is the external portion of the posterior root, has in no case shown any appreciable number of degenerated fibres (fig. 7).

There remains one other anatomical point to be considered. We have seen that in all the segments of these cases the degeneration of these root fibres has always commenced at the point where they have entered the central nervous system, and the question arises, is there any anatomical condition here which will assist in the explanation of this constant appearance? Obersteiner (5), in his "Anatomy of the Central Nervous System," describes the histological structure of the posterior root, where it enters the cord, as follows: "At the point where the posterior root pierces the pia mater it is constricted, and sometimes to a marked extent." "This reduction in size takes place entirely at the expense of the myelin sheath. Consequently, when stained by Weigert's method, the root at this point remains colourless, and a bright band, usually convex on the outer side, is seen traversing the root at the point of constriction."



Photomicrograph. Sanger-Shepherd three-colour process.

Having noticed the regularity with which the Marchi reaction has begun on the central side of this constriction, only a few droplets having been seen on the distal side, and these immediately near the ring, we have examined this region in many posterior roots by the Kultschitzky-Wolters and the van Gieson methods in serial section. (See coloured plate.)[1]

The former method shows the blue-black myelin sheath of the intramedullary portion of the posterior root fibres separated from that of the fibres in the root itself by a clear zone, in which no stain is visible.

By the van Gieson method, which stains the myelin yellow, and the axis cylinder and the fibrous structures red, we find that at the position of the clear ring mentioned above, the yellow colouration with the picric acid is absent, and, instead, this zone is stained a deep red. We also see that the deep red of the acid fuchsin is much more marked in the root outside the cord than it is in the intramedullary portion, while with the yellow stain of the picric acid the reverse is the case. These appearances depend on the histological structure of the root fibres outside and inside the cord. The fibres, in their extramedullary portion possess a sheath, the neurilemma, outside the myelin sheath, which is wanting on the fibres in their intramedullary course; and the myelin sheath itself is much thinner around the fibres on the ganglion side of the ring. At the ring of Obersteiner the neurilemma is supposed to blend with the pia mater.

We have been doubtful sometimes whether the myelin sheath is entirely absent at this point, but, with the methods at present at our disposal, its absence seems to be probable.

At this point, therefore, there is a change in histological structure, which consists of a loss of the neurilemma, and an absence, in all probability, of the myelin sheath. The appearances produced by the Marchi method support this view, because no black reaction has been found at this point.

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[1] We have to acknowledge the assistance of Dr. Bythel (Manchester) in the preparation of this photograph.

The anatomical distribution of the degeneration shows us that we have here to deal with a system lesion of the sensory neurons of the first order, which springs from a definite point in every instance, and affects, to a varying extent, the main trunks and branches of the posterior roots in their intramedullary course.

After repeated examinations of the posterior roots, between the ganglia and the cord, we can state that no appreciable degeneration or other morbid change has been discovered by the methods of Marchi, Wolters, or van Gieson. It is true that by Marchi's method a few fine isolated drops have been seen, but it is impossible to associate such an appearance with the mass of degeneration in the cord, in the relation of cause and effect. No matter whether the amount of the degeneration in the cord be great or small, nothing more than a few isolated droplets has been found in the roots. (This statement holds good for the anterior roots also.)

It might be objected that the degeneration of the roots had been present at some former period and had undergone absorption, but the coincidence would be indeed unique if the absorption of the altered myelin had advanced to the same point in all the affected segments in our cases, as well as in those of other observers. We must remember also that the age of the degeneration varied in the different cases, and even in the different segments of these cords. Further, the methods of Wolters and van Gieson showed neither atrophic nor sclerotic changes, which they certainly would have done if degeneration had been present.

The absence of the degeneration in the roots makes it at once apparent, therefore, that the intramedullary lesion is not secondary to destruction of the ganglion cell or to disease of the root itself, and we are left with the only other alternative, that the sensory fibres have been attacked somewhere in their intramedullary portion, and we maintain that it is at the point where they enter the cord that they have been attacked by some noxious agent, which has been sufficiently strong to produce a degeneration which has then spread in a centrifugal direction. This view is

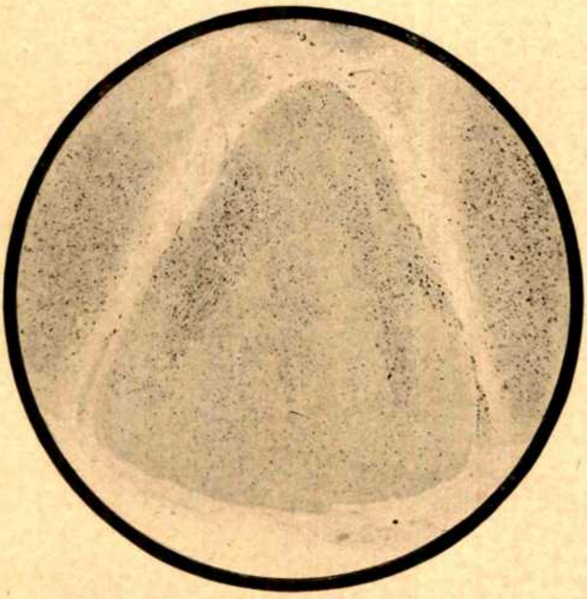

FIG. 4.
Eighth dorsal. *Photomicrograph.*

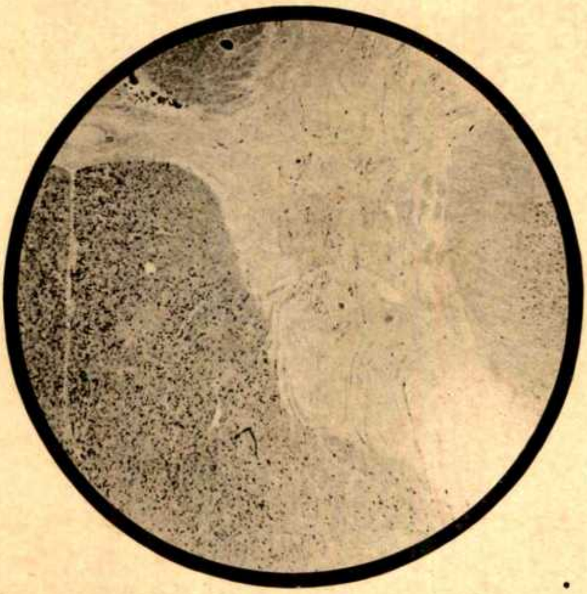

FIG. 5.
Second lumbar. *Photomicrograph.*

directly opposed to that of Nagoette (6), but to this point we shall refer later.

That noxious agents can bring about the degeneration of nerve fibres in any part of their course has been well established, both by experimental and pathological work, and we need not here enter into a detailed account of these primary degenerations of the cord. They have been repeatedly produced experimentally, and have been constantly found in the various anæmias, cachexias, intoxications, and infections, *e.g.*, chronic uræmia, diabetes, tumours. In short, they may be caused by the action of any poison on the nerve fibre, or by a disturbance of its nutrition. The condition is characterised by a progressive atrophy of the myelin sheath, while the integrity of the axis-cylinder is maintained for some considerable time.

But a primary degeneration may pass on to become a secondary one if the action of the poison be prolonged and the axis-cylinder attacked, so that, given a toxic agent in contact with a nerve, there is always a risk of secondary degeneration. And, according to Lugaro (7), the stronger or more localised the action of the toxin, the more often does secondary degeneration take place, whereas a diffuse and weaker toxic agent tends to produce a purely primary change.

Lesions very similar in distribution to those in our cases have been observed by Bruce (8), Homén (9), Nageotte (6), Williamson (10), and Erbslöh (11).

The degenerations of the posterior columns in general paralysis and in cases of cerebral tumour, described by Homén (12), coincide in origin and distribution with those which we have described. He has found that the degenerative process begins just inside the margin of the cord, and affects the exogenous zones principally and the collaterals, especially those concerned in reflex action. The lesion is more marked in the cervical and lumbar legions.

Another observation which he has made, and which we have been able to confirm, is that the changes in the posterior columns of the higher segments are in direct continuity with the changes in the lower part of the cord, after making due allowance for the passage of degenerated fibres into the gray matter, and for the influx of fresh degeneration at intermediate levels. This point we have emphasised above, and we have also drawn attention to the modification in form which the degeneration in the entry zone exhibits when the fibres which have entered at any single root level are more affected than are those which have entered in the adjacent segments.

Erbslöh (11) also has stated that it is shown by the Marchi method, that the degeneration begins where the neurilemma is lost, and that, so long as the fibres in the root are covered by the neurilemma, they show no degeneration. He also affirms that at the ring of Obersteiner-Redlich, both the axis-cylinder and the myelin sheath become disintegrated.

The recent observations of Nageotte (6) on the changes in the posterior columns in cases of cerebral tumour are very interesting. He finds that the Marchi method begins to give a positive result immediately the sensory fibres enter the cord. The exogenous system is affected, including the collaterals and the fibres passing to Clarke's column. Lissauer's area shows no Marchi reaction, nor does the extramedullary portion of the posterior and anterior roots, but a definite inflammatory focus exists at a point just central to the root ganglion.

This inflammatory focus we have never seen, but, with the exception of this, the lesion which he has described is similar to that which we have found in our cases.

According to Nageotte, the neuritis produces a Wallerian degeneration which begins at the distal extremity of the fibres and spreads backwards towards the trophic centre. In his cases the process is assumed to have reached only to the point where the fibres enter the cord.

This assumption of Nageotte is based upon his acceptance of the opinion of Ranvier, that the Wallerian process travels centripetally along the fibres, *i.e.*, from their terminals towards the point of injury. But there is strong evidence against this view in the work of Erb, Tizzoni, and Neumann (13), and in the researches of Mönkeberg and Bethe (14). These last two authors, working with the tissues of cold-blooded animals, have shown that in the peripheral portion of a divided nerve the degenerative process always proceeds

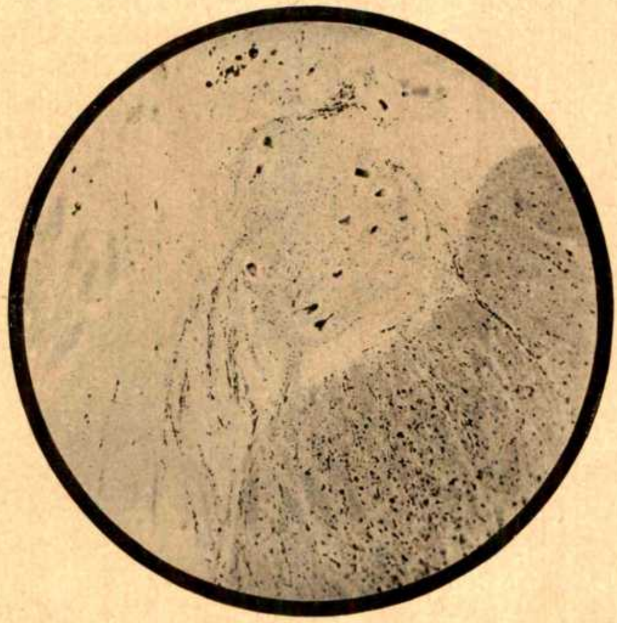

FIG. 6.
First lumbar. *Photomicrograph.*

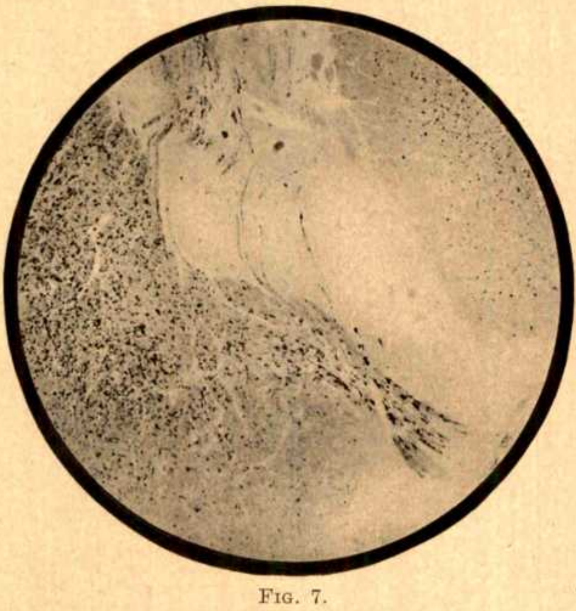

FIG. 7.
Second lumbar. *Photomicrograph.*

centrifugally from the point of injury in both the axis-cylinder and in the myelin sheath.

Moreover, we have often seen some degeneration of the fibres immediately after their entrance into the cord and in the root-entry zones, while little or no change was discernible in the collaterals in the gray matter. If Nageotte's view were correct, cases would be met with in which the Marchi reaction would appear first in the gray matter, and the degeneration would spread backwards into the entry zones, and the portion of the fibres near the margin of the cord would be left intact. As far as we can discover this has not yet been observed, and Nageotte has still to explain why the degeneration invariably reaches the Obersteiner-Redlich ring and stops there.

The integrity of Lissauer's area he attempts to explain by suggesting that the fibres of this tract are of endogenous origin, and, therefore, are not influenced by the neuritic process. Although we are in agreement with Nageotte with regard to the distribution of the intramedullary lesion, yet, in view of his histological and pathological facts, we cannot accept his explanation of the integrity of the extramedullary part of the roots and of Lissauer's zone. Why the latter should escape in acute lesions is certainly difficult to explain, but before we can accept that Lissauer's tract is composed of endogenous fibres we shall require a much stronger argument than that advanced by Nageotte. The mere fact that it does not participate in the degeneration is no proof of its endogenous origin, and we must bear in mind that the fine fibres in the peripheral nerve, which go to form this tract, have been shown by Ballance and Stewart (15) to degenerate more slowly than the coarse fibres after division of the nerve. This suggests that they possess a greater power of resistance. In view, then, of the weight of evidence which supports the theory that the secondary degeneration proceeds from the point of injury in a centrifugal direction, we regard the point at which the sensory fibres become part of the central nervous system as the focus at which these acute lesions of the posterior columns commence. We are convinced that such lesions are not the result of injury to the ganglion cells or roots, and we have not been able to discover any changes in the meninges at the Obersteiner-Redlich ring, which could act mechanically on the fibres. But, from our investigations, we have arrived at the conclusion that these degenerations are caused by some noxious agent which circulates in the lymph and acts on the nerves at the point where they lose their neurilemma sheath.

That this point in the path of the sensory protoneurons may be regarded as one of special vulnerability is shown by the study of the acute and chronic system lesions, as well as by the recent work done to determine the direction of the flow of the lymph stream in the spinal roots and in the posterior columns of the cord.

Our knowledge of the course of the lymph stream in these regions has been added to considerably during the last few years. According to d'Abundo (16), and Guillain (17), the lymph in the posterior columns pursues an ascending course, and, at the same time, passes forwards towards the central canal.

That the lymph spaces of the pia covering the posterior part of the cord communicate with those of the posterior columns is shown by the diffusion of coloured fluids (Guillain). In their last work, Marie and Guillain (18), assert that there is a common lymph system—distinct from the other regions of the cord—for the posterior roots, the posterior columns, and the pia covering them, and that the lymph is not contained in vessels, but flows freely in the intervals between the fibres and the other structures.

Homén's experiments (19) show that the lymph in the peripheral nerves ascends to the cord, and that toxins are transmitted more readily by the posterior than by the anterior roots, so that the posterior columns are more exposed to irritants than the other parts of the cord.

This upward passage of the toxins in the sheath of the spinal roots we have ourselves observed in a case of staphylococcic infection of the tissues around the spinal ganglia of the cervical region (20). From this focus the toxins spread up the roots without injuring them, but at the point where the fibres entered the cord the Marchi method gave a

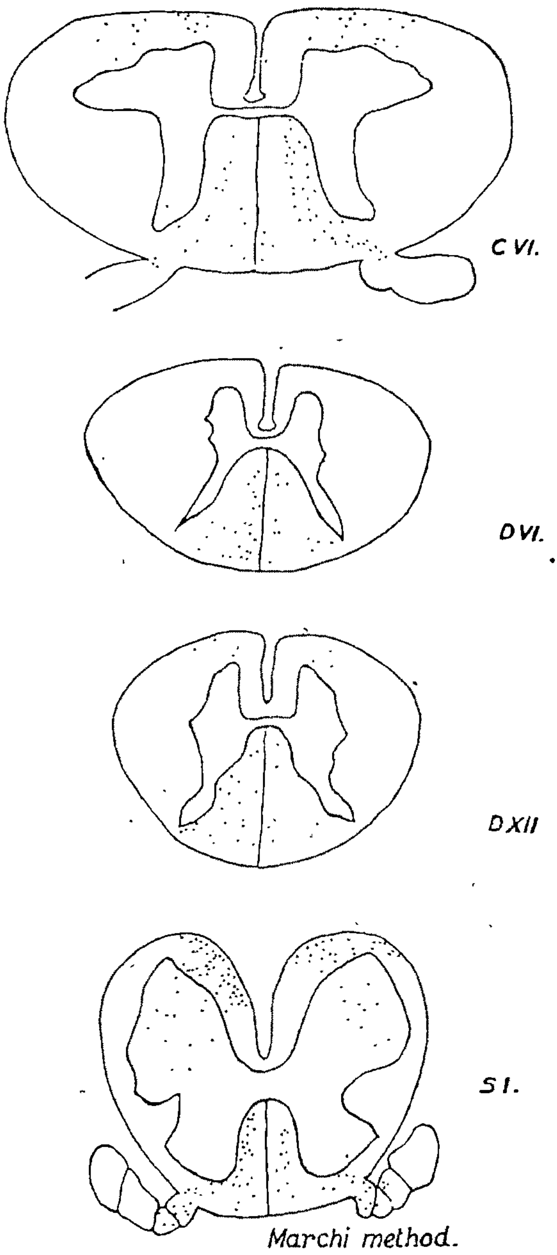

FIG. 8.

Drawn by Dr. Le Fanu.

positive reaction, which was continued into the column of Burdach.

It is possible that the existence of an ascending stream of lymph from the large nerve trunks into the posterior columns may help to explain why some degenerative lesions of the sensory fibres attain their maximum in the cervical and lumbar regions. One of our cases certainly tends to support this result, because, with the existence of bilateral bedsores, that on the left buttock being the more severe, we found that there was more degeneration in the corresponding left posterior column in the lumbar enlargement; while, with a similar lesion of the right elbow, there were many more degenerated fibres in Burdach's column on the right side in the cervical region (fig. 8).

But in cases of diabetes, cerebral tumour, general paralysis, &c., in which no peripheral toxin producing focus exists, and yet the degeneration of the entry zone is more marked at the enlargements, some other factors must come into play in the determination of this peculiar distribution.

We are, however, certain of this fact, that whether the cerebro-spinal lymph is toxic from a peripheral source or from a general intoxication, the sensory fibres are prone to give way at the point where they lose their neurilemma sheath.

Speaking generally, the dorsal nerves are less affected than the cervical or lumbo-sacral nerves, and, moreover, the number of degenerated fibres in adjacent roots may differ considerably, and thus, as we have stated before, the systemic character of the lesions may appear to be modified at different levels.

The initial change in the injured nerves is a primary degeneration, but owing to the intensity of the poison and its continued action at the vulnerable point the axis-cylinder is early attacked and a secondary degeneration rapidly ensues. At this stage the process, spreading centrifugally along the nerves, can be designated by the term secondary degeneration, because the implication of the axis-cylinder cuts the intramedullary part of the fibre off from its trophic centre.

The first portion of nerve left uncovered by the neurilemma suffers initially, but with the toxic lymph passing upwards and forwards the neurons are probably injured at other points in their course, *e.g.*, in the entry zone. To take an example from the case, which is given in detail, it is evident that the fibres of any given lumbar segment must have come in contact with toxic lymph not only in the segment to which they belonged, but also with the lymph entering the segment immediately above.

It is obvious, therefore, how this degeneration differs from the Wallerian change, which is seen in the peripheral portion of a divided nerve.

But the process is different only in development; the end is the same: a fact which is proved by the involvement of the collaterals and terminals in the gray matter, and by the degeneration of the long fibres in Goll's column. Here one sees only the centrifugal extension of the secondary degeneration.

Now, given a secondary degeneration of a system of fibres in the central nervous system there never occurs a regeneration by means of which new nerves are formed. Complete regeneration is said to occur in the cord of the lower forms of animal life, *e.g.*, in amphibia; but Spiller and Frazier (21) have never found the slightest trace of it in dogs. Gradual absorption of the degenerative products, with progressive sclerosis, must be the end of these system lesions.

This is frequently found in the cords of general paralytics in all stages of development.

A sclerosis in the posterior columns is exactly what one finds in early cases of tabes dorsalis, so that the degenerations under consideration acquire more importance when we apply them to the study of this disease.

But the point of origin of the tabetic process is still in dispute. The old theory that the fibre degeneration is secondary to ganglion cell changes has been refuted by the works of Marinesco (35), Thomas and Hauser (22).

Vulpian, quoted by Homén (2), considers the intramedullary lesion a Wallerian degeneration, caused by a neuritis of the posterior roots.

Marie and Guillain (18), after taking into consideration certain facts concerning the posterior lymphatic system—to which we have referred above—state that tabes is a lesion of this system, most probably of syphilitic origin.

In 1897 Philippe (3), in his article on the subject of tabes, said that to describe it as a sclerosis of the posterior columns and roots was insufficient; in fact he considered the interstitial lesions to be entirely secondary. His examination of the cords of early and advanced tabetics led him to the conclusion that the lesion of tabes begins as a primary parenchymatous degeneration, which attacks the medium length fibres first, and that this degeneration commences in the roots or at the point where the fibres enter the cord. The roots, in his cases, were certainly diminished in size, but he adds that this diminution was due to an atrophy of the fibres, that the interstitial tissue was normal, and that the interannular nuclei were not increased in number.

The atrophy of the roots shows, however, that he was not dealing with an early stage of the lesion.

Gowers (23) also has found that in cases in which the cord was much affected, the roots showed wasting of the nerve fibres and only slight increase of connective tissue.

The importance of a lesion of the posterior roots as the starting point of the degeneration in tabes has been much developed by Nageotte (24). He attributes the degeneration of the fibres in the posterior columns to a transverse interstitial radicular neuritis, which is an exaggeration of a diffuse meningitis, and which attacks the posterior roots close to the ganglion. This begins as a perineuritis, spreads inwards, and gives rise to an endoneuritis. At the seat of inflammation the injury to the fibre shows itself as a local atrophy, and although this is not sufficiently severe to kill the fibre, yet its presence produces a lowering of vitality, and induces a degeneration, which, he asserts, begins at the peripheral end of the neuron and spreads backwards towards the cell of origin. In course of time this degeneration reaches the seat of the neuritis, and then its starting point can no longer be distinguished.

Nageotte's theory cannot hold for all cases of tabes as this local neuritis is not a constant factor, nor even in advanced stages of the disease does one constantly find changes in the extramedullary part of the root at all comparable—in kind and degree—to those situated within the cord. In fact, there is sufficient evidence to show that often in the early stages of the disease no appreciable changes can be demonstrated in the roots, and that when they do appear in the later stages, they not only are greatly exceeded by the intramedullary lesions, but they also differ in character from them.

Thomas and Hauser (22), while ascribing the fundamental lesion of tabes to a neuritis of the posterior roots above the ganglion, admit that the lesion is more a distrophy than an inflammation; nor can the atrophic non-inflammatory condition of the roots in Philippe's cases be overlooked (*loc. cit.*).

Williamson (25) also, in 1897, published the record of a case of tabes, which had lasted for three years, in which the sclerosis in the posterior columns of the cord was well marked, while in the posterior roots a negative result was obtained by the Marchi and Weigert methods, and there was a doubtful increase in the quantity of interstitial tissue present. In any case, there were no lesions in the roots which could have produced the marked sclerosis in the posterior columns.

We have in cases of marked sclerosis of the root-entry zones failed to find sclerosis in the roots sufficient to account for the intramedullary lesion. This, together with the fact that numerous authors have observed extensive disease of the exogenous system of the posterior columns independent of root degeneration in conditions other than tabes, shows that changes in the extramedullary part of the sensory protoneurons are not essential for the causation of the cord lesions, and when present are secondary.

All these theories, excepting that of Nageotte's, have been based upon the study of tissues taken from tabetics in whom the disease had reached a considerable degree of chronicity, and we hold with Philippe (3), that under such circumstances it is impossible to determine exactly where the degeneration of the sensory protoneurons has commenced, owing to the secondary changes induced by the degeneration of the nerve fibres. It is the absence of these secondary changes which make the study of acute system lesions in the posterior columns so profitable, because we are able to locate accurately the starting point of the lesion and to draw important deductions.

It seems to us that there is no valid reason why the tabetic process should not begin at the same point. In fact, there is a certain amount of direct, as well as much indirect, evidence that it does so, because in early cases the changes found in the posterior roots are insufficient to account for the intramedullary lesion.

Homén (2) has summarised the subject of the pathogenesis of tabes in so thorough a manner that his remarks are well worth giving at some length. In early cases he has found that the process begins in the intramedullary part of the fibres, and the first changes are parenchymatous. He admits the difficulty of determining precisely where the process begins, but sometimes, quite at the commencement of the disease, the changes are confined to the intramedullary part of the root, and often to the point of entry into the cord or close to it. He suggests that the posterior columns are specially vulnerable because in cases of increased cerebral pressure, and in various cachexias, the intramedullary portion of the root fibres is alone affected. To account for this vulnerability he suggests that these fibres are more open to attack by their numerous bendings in the cord, by their passage through the pia where they may be subjected to mechanical injury (Obersteiner and Redlich), and because toxins pass upwards more readily by the posterior roots. At the same time he fully recognises the importance of the loss of the neurilemma.

The degenerative process having once been established, the further spread in the already weakened neuron takes place, (1) by the continued action of the original poison on the nerve, carrying on the degeneration; (2) by the production of a secondary degeneration in the distal part of the neuron (either by a direct spreading along the fibre or as a consequence of disturbance of the trophic interdependence in the different parts of one and the same neuron)? (3) by the simultaneous action of both these factors.

The further development consists in the involvement of new neurons in the same root and gradually of other roots.

Gradually the neuroglia, vessels, and lymph channels are affected by the substances set free by the destruction of nerve fibres, and in the late phases the lesion spreads into contiguous areas with the result that the systemic character of the lesion may be somewhat altered. Homén refutes the compression theory of Obersteiner and Redlich on the ground that in early cases meningeal changes are absent.

Homén's view as to the point at which the degeneration starts has been long held by Gowers (23), who found that the sclerosis was most dense in the part adjacent to the posterior cornu, through which the posterior fibres pass, and near the surface of the cord.

If we accept that tabetic lesions are primarily parenchymatous and begin at the point to which we and others have drawn attention, and consider in this connection the results obtained by Köster (26) after division of the posterior roots, then the reason why these should show no change in early cases of tabes will be evident, as also the reason why—as the disease progresses—this part of the sensory protoneuron should only undergo alteration slowly, and never exhibit changes equal in degree to those of the intramedullary part.

The accepted view that division of the posterior root produces no alteration of the spinal ganglion cells must be rejected after Köster's experiments. He has shown that division of the posterior roots causes a reaction in the spinal ganglion cells—much later than after division of the peripheral process—which induces in some cells a simple atrophy. and in others complete degeneration.

As a consequence of death of some cells the Marchi method gives a positive reaction between the point of division and the ganglion, and with simple atrophy of other cells—without any structural alteration—atrophy of the fibres must follow.

Degeneration of the axis-cylinder at the cord margin

—Erbslöh (11)—is the equivalent of Köster's experiment, with this difference, however, that in tabes all the fibres are not attacked and do not degenerate at the same period. A few are first affected, and the disease more or less gradually spreads to others. The fibre degeneration and atrophy in the posterior roots as a result of reaction in the spinal ganglion cells must, therefore, develop much more slowly than after experimental division, and, from what we know of the results of division of the peripheral process of the cell, the minority of cells will undergo complete degeneration, while the majority, recovering from the reactive phase, will remain permanently atrophied. Such seems to us a reasonable explanation of the comparative integrity of the posterior roots, even when the entry zones in the cord are sclerosed.

So far, in this paper, we have only dealt with what appears to us to be the exciting cause of posterior column lesions, and also with the anatomical structure of the fibres at the vulnerable point, which, we consider, is of great importance in their production.

We have also mentioned that in the cords which we have examined, the fibres in the cervical and lumbo-sacral enlargements were affected more than these in the dorsal region; and, moreover, in a given root, only some of the fibres had been attacked.

This has been seen in various cases of general intoxication, and it holds good also in tabes, and it is this fact which determines the varieties of syndromes, which are met with in the early stages of this disease.

This selection depends, in all probability, on some developmental anomalies, or other predisposing causes, which have rendered some fibres more liable to degenerate under the action of a poison, rather than to any "selectivity" on the part of the poison, as has been so often suggested.

With regard to the predisposing causes, there can be no doubt that the most important is syphilis. But, although it must be admitted that syphilis plays a very important rôle in connection with lesions of the central nervous system, the opinion that it is the direct exciting cause is not so strongly held as it was a few years ago, and we find that

Homén, Cassirer (27), and many others now speak of it as "the most important predisposing cause." In this connection Gowers (23), while recognising the importance of syphilis as a factor in the determination of the disease, has suggested that there is a special liability to degeneration in the elements which suffer.

Moreover, many cases have been published in which syphilis could be excluded, and the lesions of the posterior columns has been attributed to trauma, cold [von Leyden (28), Windscheid (29)], malaria, alcohol, influenza [Maurice Faure (30)], saturnism, pellagra, ergotism [Pansini (31)].

Of considerable importance also are the developmental anomalies, which are commonly seen in the spinal cords of those who suffer from spinal diseases. These anomalies, which are produced in intra-uterine life, may have been insufficient to give rise to any symptoms; but, according to many authorities, they indicate a diminished resistive power of the fibres.

Rebizzi (32) has described a case in which he found a heterotopia and a neuroma in the posterior columns of a cord, and there were present signs of an incipient tabes also. We, too, have seen developmental anomalies in the posterior columns and amongst the cells of the posterior root ganglia in general paralytics and imbeciles.

But it is also possible that the injury to the nerve fibres has been so slight that it cannot be detected by the ordinary means of investigation, but yet it may have been sufficient to produce a lowered power of resistance. Thus, we find Sciuti (33) stating that "the lesion in tabes consists of changes in fibres which started with some defect"; and Homén (34), in his article on Friedreich's disease, said, "we have to do with a weakened or anomalous condition of certain neuron systems, whose resistance against usual demands is lowered." Similar weakened conditions may be produced by the acute infective diseases, which are so common in early life, on the one hand, and by prolonged strain on the other hand.

Such, then, are the developmental anomalies and predisposing causes which assist in the determination and localisation of the lesions of the spinal cord, and which must be taken into consideration with the other causes and conditions, already referred to in this paper, when we attempt to explain the pathogenesis of these lesions.

But granted a hereditary or acquired weakness of the sensory protoneurons and a determining exciting cause, there still remains to be explained the fact, that intramedullary system lesions of these fibres begin constantly at the vulnerable point.

We consider that it is the loss of the neurilemma which renders the intramedullary portion of the sensory protoneuron more open to the attack of noxious agents. The neurilemma affords to the extramedullary part a protective influence which, in all probability, is more vital than mechanical; a view which is supported by the observations of many who have studied nerve regeneration.

Tabes, then, is a system lesion, which begins as a parenchymatous degeneration of the sensory protoneurons starting at the point where the neurilemma is lost. We thus agree with Gowers (23) when he says that "in system diseases the primary change is, as a rule, in the nerve elements and the overgrowth of interstitial tissue is secondary."

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THE FALSE LOCALISING SIGNS OF INTRACRANIAL TUMOUR.

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THIS communication is based upon the results of 161 consecutive cases of intracranial tumour which have been examined in the Pathological Laboratory of the National Hospital during the years 1894-1904. The examinations were made by Dr. Colman, Dr. Risien Russell, Dr. Batten, Dr. Farquhar Buzzard, and by the writer of this paper.

The majority of these cases came under the writer's personal clinical observation. His best thanks are here expressed to his colleagues upon the staff of the National Hospital, to Dr. Colman, and to Dr. Farquhar Buzzard, Pathologist to the Hospital, for their kind permission to make use of the clinical and pathological notes upon these cases.

The object of this paper is to show :—

(1) That local signs appearing late in the course of intracranial tumour, where general signs alone have pre-existed, are often of false portent.

(2) The relative frequency with which local signs have been due in this series of cases to the presence of vascular lesions, meningitis, hydrocephalus, local spreading œdema of the brain, secondary deposits of new growth, and posterior degeneration.

(3) That the absence of usually accepted local signs during the early days of illness in intracranial tumour is in itself a most important localising indication, confining the disease to the supratentorial region.

(4) That true localising signs at one time present may later become concealed or undemonstrable owing to the

development of other signs, and that in cases which come under observation for the first time late in the disease, diagnosis may be difficult, erroneous, or impossible.

The relative frequency with which the different parts of the brain were the seat of new growth in this series of cases was as follows:—

Frontal	24
Central	20
Parietal	7
Occipital	3
Temporo-sphenoidal	12
Centrum ovale	17
Basal ganglia	14
Brain stem and pons	22
Cerebellum	26
Other situations	16
Total	161

THE FALSE PORTENT OF LATE LOCALISING SIGNS.

The meaning which the writer wishes to convey by this heading will be at once apparent in the following examples:—

A man, aged 20 years, suffered with headache, vomiting, and optic neuritis. Seven months after these symptoms appeared he was totally blind from post-neuritic atrophy. In the fifteenth month of his illness localising signs began to appear. In the seventeenth month his condition was as follows:—Paralysis of left external rectus. Marked nystagmus with the slow movement to left. Complete nerve deafness of left ear. Left peripheral facial paralysis. Left cerebellar position of head (head inclined to left shoulder, and face rotated to right). Marked head retraction during paroxysms of pain. Bilateral ataxy left > right, with lurching to left on the attempt to walk. Knee-jerks: left, normal; right, diminished. Double extensor response. His condition remained unchanged until death in the twentieth month.

The localising symptoms were apparently conclusive of a growth in the left posterior fossa involving the cerebellum and the sixth, seventh and eighth nerves. The autopsy revealed a glioma of the left prefrontal region. The cranial nerves seemed normal to the naked eye, but the intramedullary portion of the left eighth nerve showed degeneration. The cerebellum was much indented by the edge of the foramen magnum, the left lateral lobe forming more of the pressure cone[1] than the right.

In this case the entire absence of localising symptoms during the first fifteen months of illness, when several general symptoms were present, not compatible with the diagnosis of tumour in any but a "silent" region of the brain. The late development of signs indicative of a lesion in the posterior fossa was the indirect and secondary result of a tumour situated at a distance.

A child, aged 8 years, suffered with headache and vomiting, and developed, within a few weeks, well-marked optic neuritis, and signs of a left-sided cerebellar lesion. These signs persisted with exacerbations and remissions for twelve months, when convulsive seizures, commencing in the left hand and being confined to the left arm, occurred, without loss of consciousness. The Jacksonian attacks were repeated at intervals, and in some of the later attacks the convulsion became general and consciousness was lost. The child died after an illness of eighteen months. A subtentorial tumour was found, which had probably arisen in the left cerebellar hemisphere, and had extended into the right cerebellar hemisphere. The fourth ventricle was much occupied by growth, and the aqueduct was obstructed. The ventricles were considerably distended, and the dilatation was more marked in the central region of the right lateral ventricle. There was no local lesion in the right central region that would account for the Jacksonian attacks other than the thinning of the ventricular wall.

This case shows that local convulsions are not always of localising value, and that they may be symptomatic of ventricular distension.

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[1] In many cases of intracranial tumour of long duration, it is found *post mortem*, that the posterior and inferior parts of the cerebellum have become pushed down and backwards into the foramen magnum, and the medulla itself being somewhat caudally displaced, the two structures together forming a cone-shaped plug tightly filling up the foramen magnum.

False localising signs were met with in twenty cases out of 161 cases in this series = 12·5 per cent. In two cases the false localising signs were due to vascular lesions (hæmorrhage, one case and thrombosis one case), situated at a distance from the growth.

In the rest of the cases the false signs were attributable to the indirect intracranial results of intracranial new growth. They were met with in 13 per cent. of the supratentorial tumours, and only twice among fifty-four cases of subtentorial tumours.

The following localising signs occurred either singly or in combination as false signs:—

Paralyses of cranial nerves.
Hemianopia.
Jacksonian epilepsy.
Bilateral spastic paresis.
Cerebellar signs.

PARALYSIS OF THE CRANIAL NERVES.

The Olfactory Nerves.—Anosmia was only found in this series, as the direct result of local pressure from the growth, upon the olfactory tract and bulb. There was not the least evidence of the occurrence of olfactory neuritis comparable with optic neuritis, as some authors have argued. The writer has, moreover, examined a large number of cases of long-standing intracranial tumour, in which optic neuritis has caused blindness, and has not found anosmia to be more frequent among them than among patients suffering with diseases other than of the nervous system.

Hemianopia was met with in two cases.

In one case the tumour was situated in the left upper central region and was of large size. The absence of the hemianopic pupillary reaction suggested that the right hemianopia present was due to interference with the left optic tract. The hemianopia made its appearance, together with paralysis of the left fifth and right sixth nerve, four and a-half months after paralysis of the right leg had become complete. There was no *post-mortem* evidence of abnormality of any part of the optic path or of the fifth and sixth nerves.

In the second case right tract hemianopia and left trigeminal paralysis and anæsthesia appeared four and a-half years after the onset of the earliest symptoms in a case of left prefrontal tumour.

The association of right tract hemianopia with left trigeminal palsy strongly suggested that preponderant indirect pressure upon the left middle fossa by a tumour in the left cerebrum had determined the combination.

Paralysis of the third nerve was met with in two cases. It was associated with paralysis of the sixth nerve, and appeared later than the sixth nerve paralysis.

One of the cases was that of a large growth in the subcortex of the right temporo-sphenoidal lobe. Seven months after the development of optic neuritis, paralysis of the right sixth nerve appeared, followed in a few days by paralysis of the left sixth nerve. Three weeks later paralysis of the right third nerve appeared, followed in a few days by paralysis of the left third nerve.

The second case was that of a large prefrontal growth upon the left side. Nine months after the onset of symptoms the left sixth nerve became paralysed, then the right sixth, and, some time later, the left third nerve became paralysed.

The important facts which these two cases illustrate are: (1) That the third nerve palsy occurred subsequently to paralysis of the sixth nerve, and (2) that the nerves were affected earlier upon the side of the lesion.

If the paralysis of these nerves is the result of shifting backwards of the brain stem from supratentorial pressure, the effect being traction upon the nerves attached to the brain stem, in proportion as their direction is more nearly a frontal-caudal direction, then the shifting backwards of the brain stem would cause paralysis of the sixth nerve first, then of the third nerves, and lastly of the seventh and eighth, while the nerves which are transversely directed or with sinuous course would be little affected. Such traction would obviously be first manifested upon the side where supratentorial pressure is greatest, that is upon the side of the lesion.

Paralysis of the fourth nerve was not met with as an indirect effect of intracranial tumour. The course of this nerve from its origin to its point of passage through the dura mater is so sinuous that traction could hardly be exerted upon this nerve by backward shifting of the brain stem.

If it be argued that fourth nerve paralysis is difficult to determine and may have been overlooked, it can be stated with certainty that fourth nerve paralysis never occurred as an isolated occular palsy in this series of cases. Every case in which diplopia occurred was carefully investigated, and the diplopia of superior oblique palsy is so distressing as to render impossible its being overlooked in a patient who is fully conscious and who is not blind.

Paralysis of the sixth nerve was met with in twelve cases of supratentorial tumour. It was unilateral in six cases, and bilateral in six cases. The paralysis when unilateral was right-sided in four cases and in two cases left-sided. It is therefore, more common as an indirect result of intracranial tumour than paralysis of any other cranial nerve. This fact has been recorded by the majority of observers, and the explanation has been given that the sixth pair of cranial nerves are more liable to be affected as a result of general increase of intracranial pressure on account of their long intracranial course and exposed position. I am unable to accept this explanation since the intradural position of the nerve alone is exposed to the intracranial pressure, the portion between this and the sphenoidal fissure being protected by the dura mater and by the blood of the cavernous sinus.

In many cases of supratentorial tumours the tentorium is pressed downwards, and the brain stem and cerebellum are also pressed downwards. The medulla and posterior part of the cerebellum come to lie partly within the foramen magnum, the cerebellum being deeply indented by the edge of the foramen magnum so that these structures together form a conical plug which fills up the foramen magnum. This alteration in the position of the brain stem may be also demonstrated *post mortem* by the deep indentation of the crura cerebri by the free edge of the tentorium.

All the cranial nerves attached to the brain stem, with the exception of the sixth pair, are directed transversely or obliquely from their superficial origins to their exits from the subdural space. The sixth pair of nerves, however, are directly straight forwards. The shifting backwards of the brain stem can have little traction effects upon the roots of those nerves, which are directed transversely, but considerable traction effect must occur from such shifting in the case of the sixth nerves.

I wish to submit it is from this cause that sixth nerve paralysis comes to be the most frequent cranial nerve palsy, resulting indirectly from the presence of intracranial tumour.

The fifth nerve was affected in two cases, and in both of these tract-hemianopia was also present. The tumours were large and were situated in the left prefrontal region and left frontal centrum respectively. In both cases the motor paralysis appeared before the sensory paralysis.

The fifth nerve paralysis and the hemianopia probably resulted from the indirect pressure of the growth upon the middle fossa of the same side.

The sensory loss appeared in the area of distribution of the three divisions of the trigeminus in the order 3, 2, 1.

The Seventh Nerve.—Peripheral facial palsy occurred in two cases. It was preceded by paralysis of the sixth nerves. In one case a large tumour occupied the posterior horn of the left lateral ventricle; the facial paralysis was upon the left side and was not associated with paralysis of the left eighth nerve. In the second case a large tumour was situated in the left frontal region, and complete paralysis of the left seventh and eighth nerves occurred.

It may be here remarked that slight weakness of the lower face is of very common occurrence in supratentorial tumours of any situation. It is not associated with any alteration of electrical excitability. The writer has no explanation for its occurrence to put forward.

The Eighth Nerve.—Bone deafness was present in two cases. It occurred once in connection with paralysis of the seventh nerve in the above-mentioned case of frontal tumour and once without facial paralysis in a case of temporo-sphenoidal growth.

The writer's experience directly negatives the theory that auditory neuritis in any way comparable with optic neuritis occurs in intracranial tumours. Excluding these two cases, and those cases where the auditory path was directly involved by growth, he has found that defects of hearing are not more common in long standing cases of intracranial tumour than in patients suffering with diseases other than of the nervous system.

The ninth, tenth, eleventh, and twelfth nerves were not indirectly affected in any of the cases.

Speaking collectively therefore, palsy of the cranial nerves, as an indirect result of intracranial tumour, is not of very common occurrence. It was present in sixteen cases out of 161 cases, or 10 per cent. The possible fallacy of statistics was strikingly shown in this series. Of the cases occurring between 1894 and 1899 it occurred in fourteen cases out of ninety-two, equal to 15 per cent., while of the cases between 1900-1904, it occurred in two cases out of sixty-nine, under 3 per cent. One possible reason for its rarity of late years is that cases of intracranial tumour are operated upon earlier and more frequently than was formerly the case. In all the cases the cranial nerve paralysis has been a relatively late symptom.

The determination as to what period is "early" or "late" depends upon the severity of the symptoms and the rapidity of growth of the tumour. A local sign appearing three months after the onset of symptoms would be a late sign in a case where the general signs were of great and persistently increasing severity; and on the other hand, it would be an early sign in a case where the general signs were not obtrusive and increased slowly. Paralysis of the external rectus was observed as early as the sixth week in a case with severe general signs. This patient died on the tenth week, and a tumour the size of a large orange was found, the pressure signs being very well marked.

The cranial nerve palsies once appearing are generally permanent, but this is not always so. Paralysis of the sixth nerve has disappeared in two cases, and in one case paralysis of the fifth nerve, at one time complete, recovered to a great extent. In such cases it is possible either that there is some alteration in the mechanical interference with the nerves in the course of time, or that they become accustomed as it were to the changed conditions.

THE OCCURRENCE OF JACKSONIAN EPILEPSY, HEMI-EPILEPSY AND GENERAL CONVULSION IN CONNECTION WITH TUMOURS OF THE BRAIN STEM AND CEREBELLUM.

In two cases of tumours of the cerebellum local convulsion of slow spread, and confined to the arm and face, were repeatedly observed. Both were cases of long standing, and in both slight signs of bilateral spasticity were present. In the early stages of the illness the signs of cerebellar involvement were conclusive. The autopsies showed that considerable ventricular distension was present, but there was no other lesion present to account for the local convulsion.

Hemi-epilepsy was observed in one case of glioma of the pons and in one case of cerebellar tumour, while general convulsion occurred in several cases of tumour of the brain stem and of the cerebellum.

In all these cases the symptoms were of long standing when the convulsions first occurred, and in all of them the autopsies revealed considerable ventricular distension.

These remarks have reference to local convulsion of the cortical type and these must not be confused with certain local convulsions of a peculiar nature and order, which undoubtedly occur in lesions of the pons and cerebellum, and which have their origin in these structures. Such "lowest level fits" have been long since described by Dr. Hughlings Jackson. Most interesting and important observations upon local convulsion of cerebellar origin are published in this number of BRAIN by Dr. Grainger Stewart and Dr. Gordon Holmes, who have given me the privilege of acquaintance with their observations while in progress.

That hydrocephalus alone is capable of causing local convulsion is proved by the not rare occurrence of the phenomenon in cases of primary hydrocephalus.

Further, if the weighty arguments of Dr. Hughlings Jackson be followed, that every epileptic seizure has of necessity a local commencement, the distinction between local and general convulsions as local signs of organic disease of the brain becomes invalid.

It is, I think, a very commonly accepted fallacy that local convulsion is a sure sign of local gross lesion of the cerebral cortex. The commonest cause of Jacksonian convulsion is probably idiopathic epilepsy. Of the patients who have attended the out-patient department at Queen Square during the past five years, and who have suffered with local convulsion, the majority have been cases of idiopathic epilepsy, provedly so by the history and course of the malady; while a minority have been cases of local gross disease of the cerebrum.

On the other hand, patients who have attended with symptoms apparently conclusive of idiopathic epilepsy and who have later on developed symptoms of intracranial growth have been numerous.

It is submitted, therefore, that the occurrence of local convulsion of hemi-epilepsy and of general convulsion, when presenting for the first time, long after the general signs of intracranial growth have appeared, is to be disregarded as a localising sign. It is the result of secondary hydrocephalus, and the latter may result no matter where the growth may be situated within the skull. Slight bilateral spasticity is not infrequently an indication of the existence of such ventricular distension.

Bilateral spastic paresis occurred late in several cases where there was not involvement of the pyramidal tracts neither directly nor by pressure. It amounted to clumsiness of movement in the limbs, slight rigidity, increase of the deep reflexes, foot clonus, and the extensor response in the plantar reflex. In all the cases there was considerable distension of the lateral ventricles. Bilateral spastic paresis thus occurring in the late stages of intracranial tumour is to be ascribed to the cortical wasting which results from secondary distension of the ventricles and perhaps also to degeneration of the cortical elements, which the chronic partial evascularisation consequent upon slowly increasing intracranial pressure must entail.

The occurrence of bilateral spastic paresis in cases of intracranial tumour usually indicates that the growth is situated in some region where the pyramidal tracts are close together; in other words, that the growth implicates the brain stem. It is especially in connection with infiltrating gliomata of the brain stem that this sign is met with.

The series of cases here dealt with show that bilateral spastic paresis may occur as a late sign in cases of intracranial tumour wherever the growth is situated, provided that distension of the ventricles occurs: Now secondary hydrocephalus may occur with a long standing growth in any situation.

It has been already pointed out that palsy of certain cranial nerves is of frequent occurrence as an indirect result of long standing intracranial growth.

The combination of bilateral spastic paresis with cranial nerve palsies, usually a sure sign of implication of the brain stem may therefore be produced in the late stages of a growth in any situation within the skull. The determination as to whether this combination is a true or a false localising sign is sometimes difficult. The reasons for the difficulty, and the points which may serve in the discrimination, will be apparent from the following table :—

	Glioma of Brain Stem.	Late Pressure Effect.
Bilateral spastic paresis	Occurs early or late ...	Occurs late.
Degree	Slight or severe, often unilateral for a time	Bilateral.
Cranial nerve palsy ...	Almost always of nuclear type at first	Always of peripheral type
	Any nerves may be involved	Never the 4th, 9th, 10th, 11th and 12th.
	Optic neuritis often absent	Optic neuritis always severe.

The question as to whether signs significant of cerebellar lesion result from the distortion of the posterior parts of the lateral lobe of the cerebellum, in the formation of the pressure cone, in cases of long standing intracranial tumour, is one of great difficulty.

The writer's opinion is that such signs are of not infrequent occurrence. Such signs must of necessity be bilateral since both lateral lobes are distorted. Further, the signs of cerebellar disease are for the most dynamic signs, and for their elicitation it is necessary that the patient shall be in possession of his mental and physical powers. For an instance, it is impossible to detect the presence of bilateral ataxy with certainty if bilateral paresis be present, or if the patient is mentally unable to attend and obey commands. Similarly the gait and tendency to fall in a particular direction cannot be tested in a patient who cannot stand and walk, or who is blind. In the late stages, therefore, of long standing intracranial tumour the tests for cerebellar involvement are often inapplicable.

In many cases, however, of long standing intracranial growth, nystagmus is met with, unilateral or bilateral ataxy may occur, the patient's gait and attitude may suggest cerebellar disease and he may constantly fall in a particular direction and in such cases no other abnormality of the cerebellum has been found.

The writer has had the opportunity of examining a very large number of cases of cerebellar tumour and is of opinion that tumours of the cerebellum rarely fail to show the signs of cerebellar involvement, as described by Risien Russell, early in the course of the case. The same holds good for all cases of subtentorial growth that they give early localising signs, with the distinct exception of some cases of glioma confined to the pons.

If this opinion be correct, the appearance of signs indicative of cerebellar involvement, late in the course of a case where no signs of subtentorial growth had previously existed, should not be allowed much weight in local diagnosis.

The absence of the usually accepted localising signs for many weeks or months after the general symptoms have become severe is in itself of very important localising significance.

Since the supratentorial division of the intracranial cavity is roughly seven times as large as the infratentorial division, other things being equal, a non-localisable tumour has a probable supratentorial situation in seven cases out of eight. In this series the proportion of supratentorial tumours to infratentorial tumours has been rather more than three to one.

While "silent areas" form a large proportion of the cerebral hemispheres, and the possibilities of compensation are considerable in this region, below the tentorium the tracts connecting the brain with the periphery are densely grouped together and further, it appears that lesions of the cerebellum are almost always early productive of characteristic symptoms. During the last five years, of a very large number of cases of cerebellar tumour in the wards of the National Hospital, there was no case verified by operation or by necropsy that had not been correctly diagnosed during life.

It appears, therefore, that a tumour which, while giving rise to marked general symptoms, affords no localising symptoms for many weeks, may be located alone the tentorium.

The Production of Symptoms from Other Lesions Co-existing with a Neoplasm.

Meningitis.

Sir William Gowers draws attention to the frequency with which some degree of meningitis co-exists with intracranial tumour, more frequently in the immediate neighbourhood of a growth which implicates the meninges, but sometimes also existing in isolated patches at a distance from the growth. With the exception of one case of tuberculous tumour of the cerebellum, in which tuberculous meningitis supervened, inflammation of the meninges was not found in this series of cases.

Vascular Lesions.

The occurrence of vascular lesions in regions remote from the situation of the tumour may give rise to prominent localising signs which are likely to lead to a false diagnosis, as in the following case: A man, aged 39 years, was admitted into the National Hospital on July 16, 1900, under the care of Dr. Hughlings Jackson. He had suffered with headache and vomiting for two months, and he presented well marked optic neuritis. Two days before admission he had a fit, commencing upon the right side, in which he lost consciousness. On recovering consciousness he presented aphasia and weakness of the right arm and face. After admission the paralysis of the right arm and face became progressively worse. An exploratory operation in the left fronto-central region revealed an extensive softening. Subsequently the necropsy showed that a large tumour was situated on the right optic thalamus, and that the anterior branches of the left Sylvian artery were extensively thrombosed.

While it seems impossible to avoid diagnostic error in cases of this nature, yet it is advisable to bear in mind the possibility of vascular lesions in cases of intracranial tumour above middle age, and who have hard arteries.

In one case of frontal tumour a hæmorrhage the size of a walnut was found *post mortem* on the right lateral lobe of the cerebellum. It was a very recent hæmorrhage and had probably occurred just before death, for no corresponding symptoms were observed. A similar hæmorrhage into the cerebellum was found in a case of rapidly fatal hydrocephalus in an adult; again the hæmorrhage was very recent and no symptoms were referable to its occurrence.

It has been stated by many authorities that the presence of a tumour upon one of the larger cerebral arteries may cause extensive thrombosis in the region of its distribution. In this series tumours have often existed in close proximity with the larger arteries but thrombosis has not been met with. It may be pointed out that the pressure which a tumour can assert is governed by, and cannot exceed, the maximum of the cerebral arterial pressure.

Local Spreading Œdema.

The occurrence of cerebral œdema in the immediate neighbourhood of cerebral neoplasms is well known. It is referred to here as the probable cause of acute symptoms in two cases among this series; the patients, while in apparent good health, were seized with urgent and rapidly fatal symptoms.

A girl, aged 23 years, came as an out-patient to the National Hospital in May, 1897. She had been perfectly healthy till May, 1895, when she had three sudden attacks of loss of consciousness at intervals of a few days and in the third of these attacks she was slightly convulsed. For the following year she suffered with occasional attacks of giddiness associated with pallor, and it was for such attacks that she sought advice. She presented no sign of organic disease. She was treated with bromides and her attacks of giddiness became less frequent. She came regularly to the out-patient department for the next eighteen months, during which time her health was good and she continued her work. On November 12, 1898, she was apparently in excellent health. The following morning she was found to be very somnolent. The somnolence deepened and she was admitted as an in-patient into the National Hospital, under the care of Dr. Bastian, on November 14. When admitted she was evidently very ill physically and she lay in bed in a half conscious state and never moved a muscle unless roused. Early optic neuritis was the only other abnormal physical sign that could be detected. Two days later paralysis of both sixth nerves made its appearance. The optic neuritis became severe, the coma deepened, and she died on the fourteenth day of her illness. The autopsy was performed by Dr. Risien Russell. A small, hard, thickly encapsuled tumour, evidently of long standing, was found, involving the second and third left frontal convolutions and the posterior part of the orbital lobule. The measurements of the growth were 5 × 3 × 3 cms. For a considerable distance round the growth the white matter of the centrum semi-ovale was much swollen. It was much

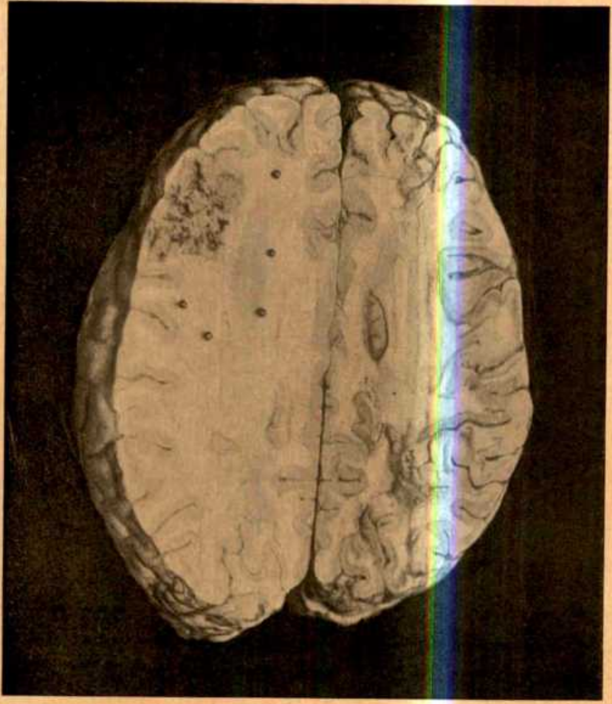

FIG. 1.

Acute œdema in the region surrounding a hard, slowly-growing tumour.

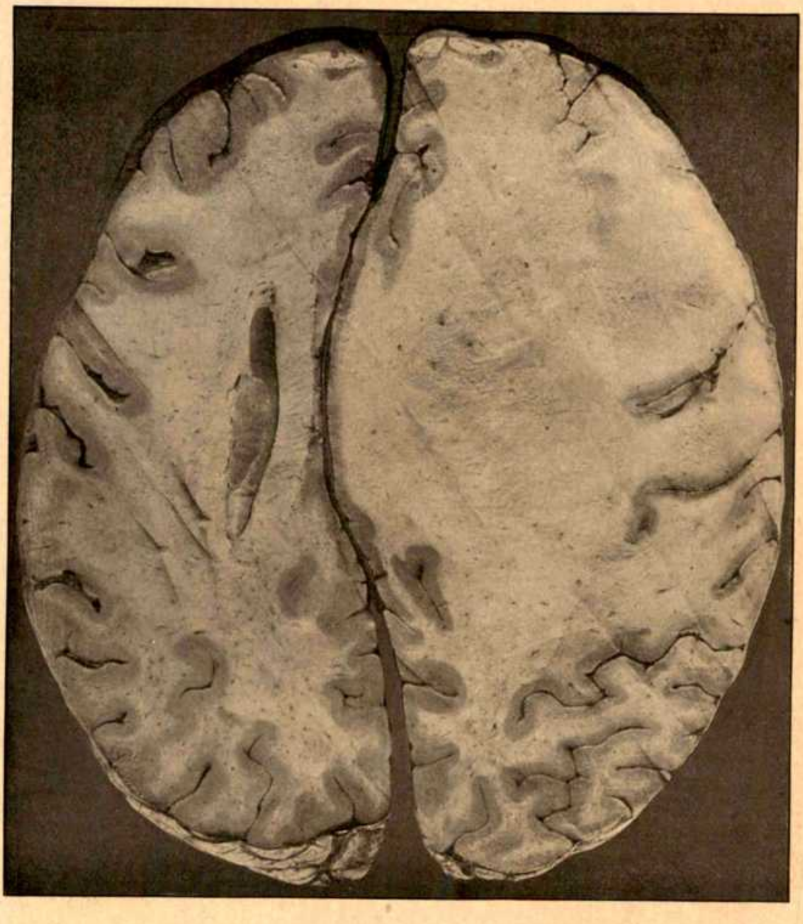

FIG. 2.

Œdema of the centrum semiovale, from a small nodule in the right prefrontal cortex, secondary to a renal carcinoma.

softer than normal and was almost translucent. Examined microscopically this swollen white matter presented marked vacuolation and lymphatic distension as the only peculiarities (fig. 1).

In this case one cannot but conclude that the epileptic symptoms were referable to the presence of the growth, and that the urgent and rapidly fatal symptoms were the result of the spreading œdema.

The second case in which symptoms were referable to spreading œdema, was one in which, as a result of primary renal carcinoma, a secondary tumour about the size of a walnut occurred in the cortex of the right prefrontal lobe. The cerebral symptoms were very urgent and death occurred in fourteen days after their appearance. There were no localising signs during the first seven days. Afterwards, some degree of hemiplegia appeared.

The right hemisphere was found to be greatly increased in size and its white matter enormously swollen even as far back as the occipital region and softer than normal and somewhat jelly-like in appearance. The tumour was sharply defined (fig. 2).

It is submitted that in this case the rapidly fatal cerebral symptoms were directly referable to the condition of œdema of the right hemisphere, and not to the presence of the growth.

SYMPTOMS WHICH MAY RESULT FROM THE OCCURRENCE OF DEPOSITS SECONDARY TO A PRIMARY CEREBRAL GROWTH.

Secondary growths are very rarely met with in cerebral neoplasms. They occurred in two cases among this series. In both cases the primary tumour was situated in the neighbourhood of the fourth ventricle and in both the growth had burst through the ependyma or the pia and was in contact with the cerebro-spinal fluid. In one case, a secondary growth was found at the junction of the anterior and posterior roots of each of the spinal nerves, from the third cervical region to the lowest sacral region, a naked eye appearance resulting of great enlargement of each posterior root ganglion. The filum terminale was the seat of many secondary growths, and its appearance resembled that of a string of beads. The symptoms referable to those secondary growth were severe shooting pains in all the limbs. Later, the knee-, wrist- and elbow-jerks were lost, and complete motor and sensory paralysis of the fifth and sixth cervical nerves on the right side occurred (fig. 3).

In the second case, a solitary secondary deposit was found upon the left first dorsal root. Right hemiplegia was present, and the patient lay in a half-unconscious state many weeks prior to death and no symptoms referable to the growth were detected.

In these two cases it was quite obvious that the secondary deposits had been planted by the cerebro-spinal fluid, and they afford important confirmatory evidence to the opinion that the main path of exit of the cerebro-spinal fluid from the intradural space is along the spinal nerve roots.

The nature of the neoplasms was that of fibro-sarcoma.

SYMPTOMS WHICH MAY POSSIBLY RESULT FROM POSTERIOR DEGENERATION.

The frequency with which degeneration of the posterior columns of the spinal cord occurs in association with intracranial tumours, no matter where these may be situated, has been pointed out by Dr. Batten and the writer, in this journal, vol. xxii., p. 473.

In some cases this degeneration is very marked and in three such cases among this series, shooting pains in the limbs, loss of the deep reflexes in the lower extremities, and ataxy of the legs, occurred, and were presumably referable to the posterior degeneration.

THE CONCEALMENT OF LOCALISING SIGNS.

Many cases of intracranial tumour come under observation for the first time long after the onset of symptoms, and, in some of these cases, localising signs, which judging from

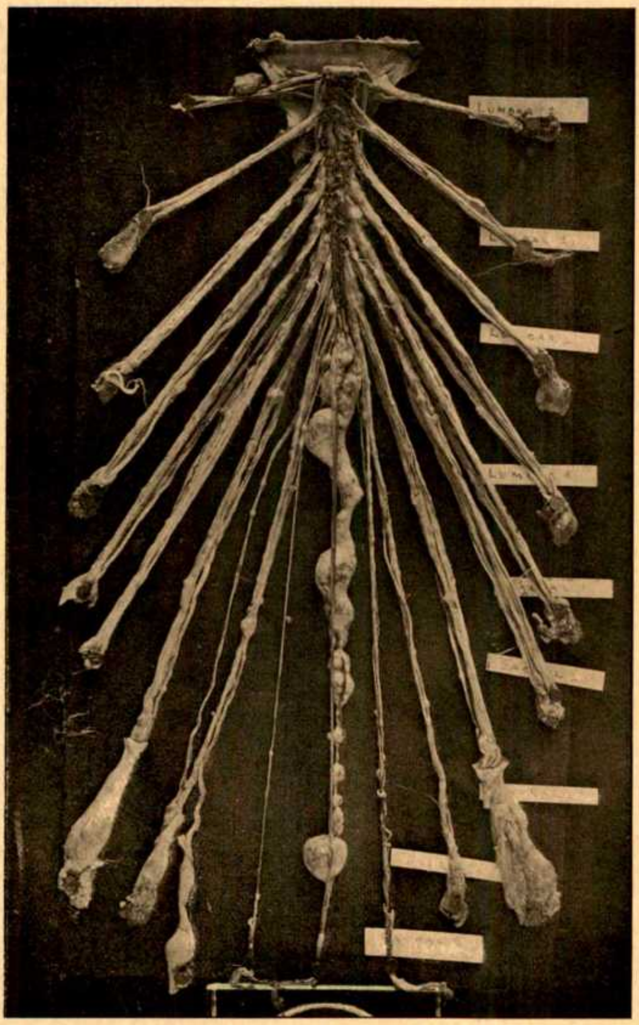

FIG. 3.

Secondary deposits in the filum terminale and root sheaths from a case of fibro-sarcoma of the cerebellum. Dr. F. E. Batten.

the situation of the lesion, must have been present in the early days, are no longer recognisable.

HEMIANOPIA.

Two cases among this series came to the National Hospital presenting no localising signs and in which optic neuritis had produced complete blindness. Careful questioning, both of the patients and their relatives, revealed no indication that hemianopia had existed while sight remained.

In both cases, the tumour directly involved and apparently originated in the region of the cuneus and hemianopia must have been present early. But this, the only localising sign of a lesion in the posterior pole of the hemisphere, had been obliterated by the optic neuritis.

CEREBELLAR SIGNS.

While diagnosis should not be difficult in the early stages of cerebellar tumour, it may be very difficult in cases seen for the first time when the disease is of long standing. The signs of cerebellar disease may be called dynamic signs. It is necessary for their proper elicitation, that the patient be in the possession of his mental faculties, and of his power of voluntary movement. He must be able to sit up, to move his limbs, to stand, and to walk, and to attend intelligently to command.

In case of long standing, as a general result of intracranial pressure or of ventricular distension, mental deterioration, physical weakness, blindness from optic neuritis, and bilateral spastic paresis may conceal the cerebellar signs more or less completely, while the occurrence of hemiplegia, or of double hemiplegia from extension of the growth into the brain stem, or from pressure upon the brain stem, effectually conceals the cerebellar signs in the region of the paralysis.

THE DISAPPEARANCE OF LOCAL SIGNS.

The local signs which result from pressure and altered anatomical relations are sometimes temporary. It has been already pointed out that indirect cranial nerve palsies may disappear in the course of time. Hemiparesis from pressure upon the pyramidal path may be similarly transient. As a tumour grows, the structural relations and directions of pressure are constantly changing and a region at one time pressed upon may subsequently be relieved. Possibly a structure may become used to the pressure as it were, and may regain its functional capacity, In two cases of glioma of the pons, however, the disappearance of a pathognomonic local sign was remarkable and difficult to explain.

Both cases, when admitted into Hospital, presented optic neuritis, bilateral spastic paresis of the limbs, much more marked in the legs and loss of lateral conjugate deviation. The last sign was indicative of interference with the sixth nucleus. Both patients lived many months and secondary hydrocephalus became manifest. Notwithstanding that the other signs became progressively more marked, the paralysis of lateral conjugate deviation became progressively less marked until some three months after admission it was no longer demonstrable. No other signs indicative of involvement of the cranial nerve nuclei appeared.

A STUDY OF THE EMOTIONS.

BY W. H. B. STODDART, M.D., M.R.C.P.

(1) The Physical Basis of the Emotions.

In the following paper I shall endeavour to establish these two propositions :—

(1) That an emotion is a sensation-complex, its component sensations being entirely derived from a complex, usually involuntary, motor response to a percept or idea of some situation or incident.

(2) That the motor paths subserving the function of this involuntary response are those of the primitive nervous system, viz. :—the cortico-rubral system of fibres and the rubro-spinal tract.

There are some psychologists who would take exception to the word "entirely" in my first proposition, and who would say that organic sensations are merely superadded to a tone of feeling which is in some way attached to the idea of the situation. Professor Titchener, for example, writes in his "Outlines of Psychology": "It is essential, then, for the formation of an emotion, (1) that a train of ideas shall be interrupted by a vivid feeling; (2) that this feeling shall mirror a situation or incident in the outside world; and (3) that the feeling shall be *enriched*[1] by organic sensations, set up in the course of bodily adjustment to the incident."

Professor Titchener makes no attempt to explain the origin of this supposed initial feeling; but, as there are several psychologists who still share his view, it becomes necessary, in the first place, to re-state the case for the opposite side, which regards an emotion as a complex of organic sensations.

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[1] The italics are mine.

The most elementary type of emotion is the feeling of pleasantness or unpleasantness which attaches to simple sensations. Introspection of these tones of feeling is very difficult, for reasons which will be understood later. For the present, it may be noted that attention to a tone of feeling necessitates inattention to the sensation by which it is aroused. Observations have been accordingly made on the physical concomitants of affection, and it has been shown by means of—

(1) *The plethysmograph*—That a positive tone of feeling (pleasantness) is accompanied by an increase of bodily volume (dilatation of arterioles), and a negative tone (unpleasantness) by a decrease.

(2) *The sphygmograph*—That a positive tone of feeling is accompanied by an increase of pulse frequency, a negative tone by a decrease.

(3) *The pneumograph*—That a positive tone is accompanied by an increase, a negative tone by a decrease of the depth of respiration.

(4) *The dynamometer*—That a positive tone is accompanied by an increase of muscular power, a negative tone by a decrease.

(5) *The automatograph* (a scientific form of planchette) —That a positive tone is accompanied by abduction of the arm, and a negative tone by adduction.

A tone of pleasantness or unpleasantness never arises without some or all of these physical accompaniments. They are the results of a general tendency on the part of the organism to reach out to that which is pleasant and useful and to withdraw from that which is unpleasant and harmful. There is little doubt that future research will discover many more movements associated with tones of feeling.

We are constantly using this phrase "tone of feeling" in discussing the nature of affection; and by retrospection, which differs but slightly from, and is in many cases the same thing as, introspection, we find that the phrase is well founded. The affective tone of pleasure or pain is a feeling or sensation superadded to the sensation which gives rise to it; and it becomes our duty to look round and see if we can discover any stimuli which may be regarded as the cause of this superadded sensation.

The experimental results obtained by means of the plethysmograph, pneumograph, automatograph, &c., supply the evidence we seek. We find that in affective states, stimuli to muscular and circulatory sensations occur in all parts of the body; and it is to be inferred that these give rise to the superadded sensations which constitute the feelings of pleasantness and unpleasantness.

From the above considerations, therefore, we learn that feelings of pleasantness and unpleasantness are due to muscular and circulatory sensations which result from complicated movements evidently reflex in nature.

Now, the tone of feeling which attaches to a percept or idea is much more complex in nature than that which attaches to a simple sensation, and it has correspondingly a very much larger number of varieties. Inasmuch as the emotional tone differs with almost every percept, idea and concept of things, people, incidents and situations, which may occur, a satisfactory classification of the emotions is practically an impossibility. The feeling of attraction towards people and things may take the form of interest, familiarity, intimacy, reverence, or love. Repulsion may take the form of dislike, disgust, antipathy, contempt, repugnance, disdain, hatred, and perhaps anger. Ideas of welfare may be associated with feelings of satisfaction, gratitude, contentment, joy, hope, or anticipation; ideas of harm with feelings of sorrow, grief, dissatisfaction, resignation, despair, fright, or horror. And if the ideas are of the welfare or injury of others, we may have feelings of gratification, gladness, envy, jealousy, regret, care, or sympathy. To all these add the emotions of misery, effort, decision, defiance, pride, shame, mirth, and the emotional tones which characterise mental operations, such as recognition, comparison, discrimination, judgment and reasoning; and the list remains yet incomplete, because our language cannot take account of the numerous intermediate and mixed shades of feeling which may attach to any one of the above-named emotions.

It has been pointed out by Professor James that an emotion consists of a number of sensations, and that these sensations are derived from the activities of certain muscles (voluntary and involuntary), and glands (sudorific, lacrymal, intestinal, &c.). The activities of involuntary muscles give rise to certain circulatory changes, such as increased or diminished frequency of the pulse, as well as to local flushings and pallors. And the more we investigate the matter, the more we become convinced that these sensations are the very essence of emotion. Let the reader conjure up some emotion, and note the various sensations he experiences therewith; then let him divest the emotional feeling of all these bodily sensations, and he will find that there is no part of the emotional feeling left. These various activities which give rise to the emotions are also responsible for the expression which indicates to others the nature of the particular emotion which is experienced.

According to this view, it is not the emotion which gives rise to the expression, but the expression which gives rise to the emotion. The question of the truth of this assertion has been appropriately referred to numerous actors: they have been asked whether they actually feel the various emotions they portray upon the stage. The best actors appear to be unanimous in the verdict that they actually feel the emotion they portray, especially when they are acting an emotional part well; and this is the reason why I have included the word "usually" in my first proposition. The experiences of the audience are in this connection no less interesting than those of the actor. When a member of the audience feels that he is being too much overcome by the sadness of the situation on the stage, he extends his trunk, assumes a smile, takes a deep breath, and surreptitiously wipes away the starting tear; by this means he dispels the emotion. And how often is an ill-timed merriment suppressed by assuming the expression, for example, of attention!

In this connection we now see why we are unable to attend to an emotion or affective tone. The particular combination of movements which constitutes the physical basis of attention disarranges the particular combination which constitutes the physical basis of other tones of feeling.

In the emotions accompanying pleasant ideas there is an increase of muscular tone and power with a tendency to extension of the trunk and abduction of the arms, an increase of pulse frequency with general dilatation of the arterioles, and an increase of the frequency and depth of respiration. In the emotions accompanying unpleasant ideas the reverse bodily conditions obtain. This much we have learned from the study of affection. But, in addition to these physical signs, there are others in the various emotions, each complex giving rise to that expression which is characteristic of the particular emotion. In anger, there is contraction of the corrugatores superciliorum, fixation of the gaze, dilatation of the nostrils, tightening of the lips, grinding of the teeth, clenching of the fists, extension of the trunk, and flushing of the face. In disdain there is contraction of the levator labii superioris alæque nasi. In suspicion there is rapid lateral oscillation of the eyes. In dissent there is lateral nodding of the head, in assent there is antero-posterior nodding. If any of these expressions be well assumed, the corresponding emotion is experienced.

It has been shown by Darwin and others that all these apparently purposeless movements are in reality survivals of movements which have served some useful purpose in the past history of the race or of the individual. In anger, for example, the grinding of the teeth and clenching of the fists are survivals of similar movements performed upon portions of an enemy's flesh; the mouth being obstructed by such a portion of flesh, dilation of the nostrils becomes a necessity for breathing purposes. The gaze is fixed upon the enemy, and the contraction of the corrugators is necessary to protect the eyes from the sun during combat.

The contraction of the levator labii superioris alæque nasi in disdain is the uncovering of the canine tooth preparatory to biting the object of disdain. The oscillation of the eyes during suspicion is the search for anticipated danger. The lateral nodding of the head in dissent is a survival of the movement of refusing the proffered breast during infancy, and the movement of assent is that of accepting the breast.

The general conclusion is, then, that an emotion is derived *e motu*, out of motion, that it is a feeling compounded of sensations arising as a result of complex instinctive movements which are, as a rule, reflexly aroused by the situation in which the individual is placed.

In attempting to establish my second proposition, I leave out of consideration those emotions which are aroused by assuming the corresponding expression. This action is voluntary, and therefore has its physical basis in the motor area.

There is evidence to show that the thalamic region plays the most important *rôle* in the development of an emotion reflexly aroused. If a patient has a lesion of one optic thalamus, say the right, and you tell him a joke, he smiles on the same (right) side of the face only; the smile does not occur on the left side. That this paralysis is not due to a lesion of the cortex or pyramidal tract is shown by the fact that the two sides of the face act equally when he *assumes* a smile. If, on the other hand, the patient has a lesion of the right Rolandic area, he smiles equally on the two sides in response to a joke; but an *assumed* smile occurs on the right side only, volitional action being paralysed on the left side.

The observation of movements of expression occurring in the limbs is a more difficult matter in paralysed patients; the physician has to rely upon an opportunity of observing the hand when the patient yawns. In paralysis of the hand, due to some unilateral cortical lesion, the patient is unable to open the opposite hand voluntarily, but if he yawns, the hand opens slightly. If, however, a patient has a lesion in the region of one optic thalamus, he can open the opposite hand voluntarily, but it does not involuntarily open when he yawns. The conclusion to be drawn from these observations is that the tracts subserving the motor element of emotion cross to the opposite side of the cord.

Now, the only bundle which crosses from the mesencephalon to the opposite side of the cord is the rubro-spinal bundle of Monakow—that bundle which, as Held and

Probst, and subsequently Buzzard and Collier, have shown, arises on the ventral side of the red nucleus, decussates in Forel's crossway with the corresponding bundle of the opposite side, and is traceable in the region of the lateral tracts as far as the sacral region of the spinal cord. It connects the opposite nucleus ruber with the ventral horn of the cord.

I therefore submit that Monakow's bundle subserves the function of the motor element of emotion. The only other tract which here deserves consideration is the thalamo-spinal tract of Buzzard and Collier. This is an uncrossed tract taking its origin deep in the thalamus and traceable as far as the lowest cervical region. My data are not sufficient to include or exclude this tract from the function of emotional reaction.

I now have to consider the cortical portion of the system of motor neurons subserving the function of emotion. For this function a system of fibres is required to connect the cortex with the nucleus ruber, and such a system has been described by M. and Mme. Dejerine. The fibres take their origin in all parts of the cortex, but especially in the parietal lobe. They reach the thalamus just above the radiations of the internal geniculate body, enter into the constitution of the fillet, and reach the red nucleus at its antero-supero-external part. These fibres are to be regarded as the upper segment of the emotional motor system. Their intimate anatomical relationship with the thalamus easily accounts for the fact that that structure has hitherto been regarded as the physical basis of movements of expression.

There is no evidence for or against the view that the cortico-thalamic and thalamo-spinal fibres may play a subsidiary *rôle* as part of the physical basis of movements of expression.

It is of considerable interest that the cortico-rubro-spinal motor system is at least the main representative of the primitive motor tract, the tract by which in the lower vertebrates all motor impulses are transmitted. It has been demonstrated by Münzner and Wiener, Boyce and Warrington, Edinger and others, that the pyramidal system of fibres does not exist in birds or in any of the lower vertebrates. In these animals the motor tract consists of cortico-thalamic and thalamo-spinal neurons only, the spinal fibres occupying the same relative position as the direct and crossed pyramidal system of mammals. In this connection it will also be remembered that in man the pyramidal tract is not myelinised until about the eighteenth month. Professor James has indicated the close relationship subsisting between emotions and instincts. Both are involuntary motor responses to sensations, percepts and ideas; and the only difference between them is that instincts bring the organism into more practical relation with the object of the idea, percept or sensation. Now, the lives of birds and lower vertebrates, and the life of the human infant until it is about eighteen months old, are practically little more than a mass of instinctive and emotional reactions; and it is not surprising to find that such reactions are, among the higher vertebrates, still dependent upon the functioning of the pristine nervous system.

The neural process which takes place when an emotion occurs is then as follows:—

Starting from the stage at which a sensation is registered in one of the projection areas, or a percept or idea formed in one of the association areas of the cortex, an impulse is transmitted to the red nucleus by way of the cortico-rubral fibres, thence to the large motor cells of the lowest level by way of Monakow's rubro-spinal (and presumably rubro-bulbar) fibres of the pristine motor system, and thence to the muscles of expression. Contraction of these muscles upon their spindles effects the transmission of muscle-sensations to the cortex by way of the ordinary sensory paths; and it is the particular combination of these sensations among themselves and with vaso-motor sensations which determines the given affective or emotional tone.

(2) THE PATHOLOGY OF THE EMOTIONS.

In this section I do not propose to deal with persistent states of depression or exaltation, but to consider morbid modifications of the emotional reaction to percepts or ideas of situations and incidents in the outside world. In the insane such emotional reaction may be excessive or deficient, the cause of this excess or defect differing in the various states with which we have to deal.

In those conditions dependent upon progressive deterioration of the nervous system, such as general paralysis, alcoholic insanity and, perhaps, epileptic insanity, the emotional reaction is excessive; hemiplegics, too, are very emotional; the most unimpressive word or gesture often suffices to induce an attack of weeping or laughter. The same may be said of imbecility, a condition in which the nervous system is ill-developed, and of states of maniacal excitement. Again, paranoiacs and patients suffering from hallucinations are usually liable to outbursts of anger and other forms of emotion. We have also to consider those patients who suffer from morbid fears.

Deficient emotional reaction, on the other hand, characterises confusional and stuporose states, senility and all forms of secondary dementia, myxœdema, and cretinism.

(*a*) *Excess of Emotional Reaction.*

The doctrine is now well established that dissolution of the nervous system takes place in reverse order to its evolution; and it has been demonstrated that the last motor tract to develop in the history of the vertebrate nervous system is the pyramidal tract. It is in accordance with this doctrine that the first motor tract to suffer in such progressive degenerations of the nervous system as general paralysis, alcoholic insanity and epileptic insanity, is the pyramidal tract; and the consequence is that in these diseases, and also in hemiplegia, motor impulses tend more and more to be transmitted by way of more primitive motor tracts *viâ* the thalamus.

Now, these are the very tracts which normally subserve the function of the emotional reaction; and so it happens that patients suffering from the above diseases react emotionally to unimpressive stimuli: their main outlet is by way of emotion-arousing tracts.

I do not know whether the pyramidal tracts are especially ill-developed in imbeciles; but it is extremely probable that they are, for the patient is unable to walk properly in many cases. The nervous system as a whole is ill-developed, and it is to be supposed that the pyramidal tracts, which are normally myelinised late, are disproportionately affected. This being so, the above principle also applies to the excessive emotional reaction of imbeciles.

In states of maniacal excitement, the excessive tendency to emotional reaction is dependent upon a different set of conditions. I have elsewhere shown reason for the belief that mania is a state in which the neurons contain some irritating body or bodies; the neurons are consequently in a permanent state of excitability.

The result of this constant state of tension of the neurons in maniacal states is that minimal stimuli provoke nervous discharge and, in the case of motor neurons, induce contraction of muscles.

The application of this principle to the emotionality of maniacal patients is as follows:—A perceptual or ideational process occurs in one of the association-areas of the cortex; all the neurons in functional communication with this area, especially the cortico-rubral neurons, are discharged: in other words, discharge takes place into the emotional regions of the nervous system.

Dr. Wolseley Lewis has drawn my attention to the fact that insane epileptics, while free in the movement of their large proximal joints, are stiff and cramped in the smaller ones. He also observes that the epileptic shakes hands from the shoulder. In these particulars, the epileptic resembles the maniac; and, from the principles which I laid down in my paper "On Motor Symptoms of Mania and Melancholia" (*Lancet*, March 5th, 1904), it is to be concluded that the neurons of epileptics, as well as those of maniacs, contain some irritating body within their substance. The motor neurons of epileptics are therefore in the same "touch-off" condition as those of maniacs, and this affords us another explanation of the excessive tendency of epileptics to emotional reaction. Of course, it may be that the patients to whom Dr. Lewis refers are cases of epileptic mania.

The emotional outbursts of paranoiacs and of patients suffering from hallucinations may be looked upon as being due to excessive perception, in contrast to those forms of diminution of emotional reaction which are due to "imperception" (*vide infra*). The laughing or weeping of a patient who has just experienced an illusion or hallucination takes place because the patient has perceived something which is not there; he has suffered from excess of perception.

Similarly, paranoiacs suffer from an excess of perception; their association of ideas is excessive, and they see hidden meanings in the most trivial incidents. A passer-by in the street blows his nose, and the paranoiac perceives the handkerchief as the cloak of a sneer or smile; the result is the emotional reaction we call anger.

Morbid fears are due to an abnormal tendency of the pristine nervous system to react to some particular percept. This tendency must, as a rule, be regarded as congenital, since it develops in people who are normally lacking in self-confidence. The tendency is, however, sometimes acquired; some illness leaves the patient devoid of self-confidence, or some unusual incident may initiate this morbid tendency.

(*b*) *Deficiency of the Emotional Reaction.*

If you tell a person a good joke, there are three possible causes for his not laughing at it: (1) He does not hear it, (2) he does not "see" it, and (3) he is preoccupied. These are the three causes of diminution of emotional reaction in the insane.

In confused and stuporose states the patient suffers from anæsthesia of characteristic distribution. In such cases perception is deficient because sensations are not satisfactorily served up to the ideational centres; and emotional reaction is deficient for the same reason that a deaf man does not laugh when you tell him a joke. It is also to be observed that in these patients there is a further reason for the loss of emotional feeling in that the muscular sense is defective, so that the patient would not experience an emotional feeling even if a slight motor reaction should occur.

Emotional defect may be due to partial or complete imperception. In its typical form, imperception is a condition in which the patient is unable to recognise persons, objects and things; and it depends upon organic disease of the ideational areas. It also occurs to a greater or less extent in cases of secondary dementia, senility, myxœdema, and cretinism. In all these conditions there is corresponding deficiency of emotional reaction.

In some cases of this kind, emotional reaction occurs; but its character is inappropriate to the occasion. For example, it frequently happens that an advanced dement laughs on being told that some relative, once dear to him, is dead.

Absence or deficiency of emotional reaction also occurs in severe cases of melancholia. Professor Ribot mentions in his "Psychologie des Sentiments" some cases of melancholia in which there was complete absence of emotional reaction; but the name "anhedonia" which he has given this symptom and the descriptions of his cases indicate that he has not quite fully grasped its significance. Cases of severe melancholia suffer from an absence, not only of pleasurable, but also of painful, emotional reactions. Such patients experience no pleasure when they think of their home, wife and family; as a rule they tell us that they have lost all affection for their friends; and when it becomes our painful duty to inform a melancholiac of the death of his nearest and dearest relative, he commonly remarks, "I don't seem to feel it."

The cause of this lack of emotional reaction is not far to seek. I have frequently pointed out that the greater part of the muscular system of melancholiacs is rigidly fixed owing to partial paralysis of cortical neurons, and Dr. Craig's observations that the blood-pressure of melancholiacs is raised indicates some fixation of the involuntary arterial muscle-fibres. It is on account of this motor and vaso-motor fixation that the motor and vaso-motor changes essential to emotional reaction cannot take place.

Katatoniac stupor is also characterised by muscular rigidity. This rigidity differs from that of melancholia in that it affects the whole of the muscular system uniformly; whereas the rigidity of melancholia mainly affects the musculature of the spinal column and of the large proximal joints. Dr. Craig tells me that the blood-pressure is also raised in katatoniac stupor. The practical point is this, that in katatoniac stupor there is motor and vaso-motor fixation as in melancholia; and it is on account of this fixation that there is complete absence of emotional reaction in this condition.

Professor Kraepelin considers that there is an absence of emotional reaction in other forms of dementia precox. I have been unable to convince myself that this is the case, and, on the other hand, I am unable to discover any reason why there should be any lack of emotional response in most cases of simple dementia precox, hebephrenia or dementia paranoides.

The conclusions arrived at in this section may be summarised as follows: Excess or defect of emotional reaction may be dependent upon excess or defect of sensation, or upon excess or defect of perception. Excess of emotional reaction may also depend upon an abnormal tendency of motor impulses to be transmitted *viâ* the pristine emotion-arousing motor tracts of the nervous system; and defect of emotional reaction may further be due to fixation of emotion-arousing musculature.

SYMPTOMATOLOGY OF CEREBELLAR TUMOURS; A STUDY OF FORTY CASES.

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(4) Relation of clinical symptoms in man to the data from experiments in animals.
(5) Abstract of notes of twenty-two cases in which diagnosis was confirmed.

INTRODUCTION.

This paper is based upon the study of a series of forty cases of tumour which involved the cerebellum, either directly or indirectly by pressure, which we have personally observed during the past two and a half years in the wards of the National Hospital.

We here express our thanks to the Medical Staff for kind permission to use these cases.

The diagnosis was confirmed by autopsy or by operation in twenty-two cases, of which we append the clinical details.

We have rigidly excluded cases in which the disease extended into other parts, or in which conditions were present which could complicate the symptom-complex. This series is divisible into two classes: (*a*) Cases in which the tumour was situated within and limited to the substance of the cerebellum; (*b*) and those in which the growth was extra-cerebellar. By extra-cerebellar tumours we mean those which lie in the posterior cranial fossa in the angle between the pons varolii and the cerebellum, compressing but not directly invading either. Such growths may arise either in connection with the ventral surface of the cerebellum, in which case they are generally gliomata and occasionally cystic, or from the adjacent cranial nerves, when they are generally fibromyxomata. These growths are circumscribed, encapsulated tumours, and easily separable from surrounding tissues. Plates I. and II. represent photograph from a typical case, and illustrate the relationship of these growths to their surrounding structures. Despite the degree of compression which the pons suffers in such cases, it is remarkable how little the symptoms resemble those of intrapontine growth. The pressure is chiefly exerted on the middle cerebellar peduncle.

For a fuller study of the symptomatology we avail ourselves of eighteen cases in which the diagnosis has not received confirmation, but whose clinical picture was similar to those we record. This we can do with little hesitation, relying on the unvarying accuracy of the diagnosis in that series.

Finally, having had the opportunity of observing thirteen cases in which the disease was removed by operation, which entailed large destructive lesions, we are in a position to discuss the positive and negative symptoms of cerebellar disease in relation to the results of experimental research in animals.

GENERAL SYMPTOMS.

Headache is an early and almost constant symptom of both extra- and intra- cerebellar neoplasms. Its character is practically identical with that associated with other intracranial growths, but its situation is often suggestive of the site of the disease. In intra-cerebellar tumours the headache is generally most intense in the occipital region, and is frequently limited to this area for long periods, and in this respect is unlike the pain complained of in any cases of supratentorial neoplasms which we have observed. The pain may often radiate down the back of the neck to between the shoulders, and indeed the patient may refer it only to this region and show a great reluctance to moving the head owing to the discomfort it causes. The next most frequent site of headache is frontal or retro-ocular, but though this may be its initial situation it seldom remains limited to this region throughout the whole course of the illness. The frontal headache was never limited to one side in our cases. In extra-cerebellar tumours the pain is almost constantly occipital, and may radiate down the neck. It may not be so marked a symptom as in intracerebellar tumours, and, indeed, in some of our cases it was rarely complained of.

In contrast to the pain associated with intracranial neoplasms in other sites the headache is, as a rule, extremely severe and remarkably constant. We have found little in favour of Luciani's assertion, that it is due to direct irritation of the cerebellar tissue or of Purves Stewart's assumption that its occasional frontal seat is due to radiation of stimuli along the fronto-pontine tracts.

Vomiting is an almost invariable symptom during some stage of cerebellar and extra-cerebellar tumours. In the former especially it is apt to be very frequent and severe, but its character is identical with that which accompanies intracranial growths seated above the tentorium. When severe it is always associated with headache.

Optic Neuritis is in our experience a constant accompaniment of intra-cerebellar neoplasms. It seems to be one of the earliest signs, to be remarkably intense, and often out of proportion to the general symptoms. Its onset is as a rule acute, so much so that vision may be considerably impaired early in the illness. Attacks of transient blindness may occur during any stage of the illness, and often herald the approach of permanent loss of vision. In the majority of the cases the intensity of the neuritis and the degree of swelling of the papilla have been greater on the side of the growth and the permanent blindness due to consecutive atrophy has resulted in that eye first. In extra-cerebellar growths the neuritis is generally early and severe, but it may be late in appearing, and in one of our cases (No. 14) the patient died before any swelling developed. The degree of swelling was independent of the side of the tumour.

The general character of the neuritis is similar to that met with in other intracranial neoplasms.

VERTIGO AND AUDITORY SYMPTOMS.

Vertigo is one of the most constant symptoms of extra- and intra-cerebellar tumours, and it is specially frequent in the former. Owing to its transient nature and the accompanying mental confusion it is often difficult to get a detailed or definite description of it from the patient. It may occur in two forms. In the one there is a definite sense of movement of self or of surrounding objects; in the other it is an indefinite sensation generally described as a "giddy feeling."

The subjective phenomena of definite vertigo need careful description. In both intra- and extra-cerebellar tumours the sense of displacement of external objects in front of the patient is from the side of the lesion to the opposite side. Thus if the tumour be on the left side the apparent movement of objects is from left to right.

In intra-cerebellar tumours the subjective rotation of self is always from the side of the lesion to the healthy side, that is in the same direction as the apparent movement of external objects.

In extra-cerebellar tumours the subjective rotation of self is always from the healthy side towards the side of the lesion, that is in the opposite direction to that of the displacement of external objects.

The direction of the subjective movements was so constant in each class of case, that we are led to regard it as one of the most serviceable diagnostic points. These forms of vertigo were very pronounced in many cases after the tumours had been removed by operation. The patient may complain of a sense of movement of self or of external objects and at the same time tend to roll over in bed, in extra-cerebellar cases towards the side of the disease, in intra-cerebellar cases away from the side of the disease. These forced movements, which are not invariably associated with subjective movement, *i.e.*, vertigo, will be discussed more fully later.

In one case (No. 18) of extra-cerebellar tumour there was also an apparent vertical elevation of objects in front of the patient without subjective movement of self.

The other form of vertigo which is met with in both intra- and extra- cerebellar lesions is quite indefinite. The patient suddenly becomes giddy, generally after some abrupt movement, his surroundings become dark, and there may be some numbness of the extremities or a feeling of utter weakness, associated with intense nausea and a tendency to fall backwards or towards the side of the lesion. Consciousness may be impaired, but is never quite lost. Objects may seem to swim or intermingle indiscriminately in front of the patient, and he may feel that without support he must fall in any one direction or at once collapse. These forms of vertigo have no localising value.

In a few cases of cerebellar lesion, both before and after operation, we have rapidly rotated the patients on their vertical axes in order to observe the vertigo which results. In normal individuals when the rotation is suddenly ceased the person for a short time feels that he is still moving in the direction of the rotation, and that his surroundings are revolving round him in the opposite direction. The eyes are at first in full conjugate deviation to the side towards which the rotation took place, *i.e.*, to the left when the patient is rotated from right to left, and regain their normal position by slow recession towards the middle line, interrupted by forcible nystagmoid jerkings towards that side. In the cases examined it was constantly found that the giddiness and the sense of rotation of self is less intense when the rotation has been towards the affected side than from it, and the deviation of the eyes is less complete and the nystagmus towards the middle line less marked. It must, however, be emphasised, that the eighth nerve, or at least its cochlear division, was affected in most of the cases so examined.

Deafness.—In considering this symptom the two classes of cases have to be rigidly separated. In our cases of extra-cerebellar tumours there was invariably some deafness on the side of the disease. The loss of hearing varied in intensity from complete deafness to relative reduction of acuity, which was always equally pronounced to air and bone conduction, its character being that of nerve deafness. In the intra-cerebellar cases on the other hand there was rarely any considerable affection of hearing. In a few cases there was slight deafness on the side corresponding to the disease, but this was never so great as in the cases of extra-cerebellar neoplasm.

Subjective Auditory Sensations.—Tinnitus is a fairly frequent symptom in extra-cerebellar tumours. We met with it frequently, and it was always referred to the corresponding ear. It was variously described as "the ringing of bells," "buzzing," or a "constant blowing or swishing noise." In intra-cerebellar tumours tinnitus was rarely a prominent symptom, and when present was vaguely described as "noises in the head," and was not referred to either ear. In this it resembles the tinnitus which occurs with intracranial neoplasms in other situations, and is probably dependent on the general increase of intracranial pressure rather than on local causes. Such subjective auditory sensations are never organised or elaborate.

CRANIAL NERVE SYMPTOMS.

Ocular Symptoms.—The majority of cases of extra- and intra-cerebellar tumours develop, during some stage of the illness, varying degrees of external rectus palsy on the side of the disease, but the resulting squint and diplopia are often transitory, though liable to recur. This paresis is as a rule greater in extra-cerebellar lesions than in intra-cerebellar, and though in the latter there may be affection of both external recti, it is always most evident on the side of the lesion. Bilateral external rectus paresis is especially met with in tumours of the middle lobe of the cerebellum which compress the floor of the fourth ventricle. The degree of weakness is in all cases very variable, but is never complete unless the tumour secondarily involves the pons varolii, or when an extra-cerebellar growth extends so far medialwards on the ventral surface of the pons as to directly compress the sixth nerves.

Cerebellar lesions do not appear to have *per se* any influence on the pupils. The size of the latter depends upon the degree of impairment of vision due to the optic neuritis or secondary atrophy, but when there is secondary hydrocephalus the pupils may be very large, though reacting briskly to light and accommodation. Weakness of the muscles supplied by the third or fourth nerves never results directly from cerebellar growths. In one case, unconfirmed by autopsy, there was weakness of the superior oblique, probably due to direct compression of the fourth nerve by a tumour, which lay in the anterior portion of the cerebellum.

Another ocular phenomenon frequently seen, though never in complete degree, is some weakness of conjugate lateral movement towards the side of the lesion. It is met with both in tumours external to the cerebellum and in those of the lateral lobes, but is generally greater in the former class.

This weakness may be manifested merely by an inability to sustain lateral deviation of the eyes, or it may be that greater effort is required to move the eyes to that side. In other patients, especially those blind from secondary optic atrophy, the eyes are often, while at rest, constantly deviated to the side opposite the lesion. Associated with this weakness of the conjugate movement of the eyes to the side of the lesion there may be relatively greater weakness of the external rectus of that side.

Skew deviation of the eyes is another ocular phenomenon which we have frequently observed after acute attacks or after operation. It consists of a downward and inward position of the eye on the side of the lesion, and of an outward and slightly upward position of the other eye. Its short duration suggests that it is due to an irritative condition rather than a destructive, though in one case it persisted for several weeks after operation, and in another, on which no operation had been performed, it was constant for considerable periods. (Plate II., fig. 4.)

Nystagmus.—This is a feature held in common by both classes of cases, and is one of the most valuable of the localising signs. It is almost invariably found in every case at some time or other. In the more chronic cases and when the symptoms have become latent it may be less marked and more variable. The typical nystagmus in cerebellar growths consists of slow deliberate jerking movements to the side of the lesion on looking in this direction, with gradual recession of the eyes towards the middle plane; in conjugate deviation of the eyes to the opposite side the movements are of smaller range, but occur in more rapid succession.

Though, as a rule, the nystagmus was conjugate, the movements of both eyes being equal in range and simultaneous, we occasionally observed a greater range of movement in the eye homolateral to the disease.

The nystagmus is always more marked on lateral than on vertical movements of the eyeballs. In a few cases on looking up there was irregular and coarse nystagmus often composed of rotatory movements of both eyeballs, but chiefly of the homolateral, upwards and to the side of the lesion.

Occasionally on convergence both eyes, but more frequently the homolateral alone, may jerk irregularly to the side of the lesion.

We have only observed spontaneous nystagmus in cases in which there was a marked tendency to conjugate deviation of the eyes, especially for a few days after operation. The movements were generally irregular, with a tendency to jerking of the eyes to the side of the disease and gradual recession towards their natural position.

N. Facialis.—Paresis of the facial nerve of the same side as the disease frequently occurs. It is of peripheral type, but in intra-cerebellar tumours is always slight and often variable and inconstant. As extra-cerebellar growths seem to have arisen from the flocculus or in connection with the auditory nerve, the degree of paresis in these cases has been as a rule greater and occasionally became complete, with reaction of degeneration in the later stages of the illness. In others, however, the facial paresis was very slight, or even absent when they first came under observation. In some cases of unilateral disease there may be facial palsy on the contralateral side, and it is then of the hemiplegic type owing to compression of the upper facial neurones in their passage through the pons. We have never observed facial weakness corresponding to the paresis of the homolateral limbs in cerebellar disease.

Twitching of the homolateral side of the face occasionally occurs in cases of extra-cerebellar tumour, presumably owing to direct irritation of the nerve.

N. Trigeminus.—Affection of either the motor or sensory divisions of the trigeminus occurs occasionally in intra-cerebellar tumours, more frequently and in greater degree in extra-cerebellar. In both classes the sensory root suffers more often than the motor. There may be only pain or paræsthesia (numbness, tingling, or a drawn feeling) on the corresponding side of the face, or definite sensory loss of variable degree. In the latter instance the corneal reflex may be diminished or lost and neurotrophic affection of the cornea may result. In these cases the sense of taste is frequently diminished or absent on the anterior two-thirds of the corresponding side of the tongue.

IX., X., XII. Cranial Nerves.—Affection of the functions of these cranial nerves is rarely if ever met with in intra-cerebellar disease, but in some cases of extra-cerebellar tumour there was weakness of the same side of the palate, which on movement is drawn to the opposite side, and less frequently of the vocal cords and tongue. This was due, as confirmed in several autopsies, to direct pressure on these nerves by the growth, but only in the later stages of the illness did the weakness become pronounced. As a result of these palsies articulation became indistinct, slurred, or nasal, often scarcely intelligible, and even phonation was considerably impaired. The patients had increasing difficulty in swallowing, and as consciousness became clouded nasal feeding was required. Deviation of the tongue to the opposite side was occasionally observed in cases of extra-cerebellar tumour due to the pressure on the pons varolii being exerted on the central hypoglossal connections above their point of decussation. It was in these cases that some spastic weakness of the contralateral limbs existed.

MOTOR SYMPTOMS.

In making observations on the motor system special attention has been paid to the following points: (1) The tone of the muscles; (2) the power of the various movements; (3) the co-ordination; (4) the attitude of the patient; (5) the gait. In analysing the cases it will be seen that in some of the intra-cerebellar cases the onset was recent and acute, while in others slow and gradual, and in many of the cases operated on there followed as a necessary sequence destruction of various portions of the cerebellum. Thus we have had the opportunity of observing the motor system: first, in acute intra-cerebellar growths; secondly, in chronic intra-cerebellar growths; and thirdly, of noting the immediate and late effects of extensive or more limited destructive lesions of various regions of the cerebellum which resulted from operative interference.

Paresis.—One of the most definite symptoms of unilateral cerebellar tumour is deficiency of power in the limbs of the same side. This hemiparesis is often considerable when compared with the power of the opposite limbs; in some cases, however, it is absent. It may be said to be uniform as far as the distal and proximal muscles of the extremities are concerned. This muscular asthenia must be a direct effect of the cerebellar lesion ; as it is homolateral to the side of the disease, it cannot be ascribed to any effect, direct or indirect, of the tumour on the cortico-spinal system, and in cases which terminated fatally no degeneration of these fibres could be found on microscopical examination.

Further, the character of the weakness, the absence of any organic rigidity and the normal state of the superficial reflexes, are strongly against the existence of any interference with the functions of the pyramidal tracts.

The weakness is relatively greater in cases of acute onset, and especially after extensive destruction of cerebellar tissue entailed by operation. In the latter class of cases there is often a condition of general muscular asthenia, but there can never be any doubt as to the greater homolateral paresis. The contralateral limbs very quickly regain their full power, but their fellows may remain definitely paretic for a considerable period. As regards the distribution of the weakness, there is a very considerable paresis of the trunk muscles, especially of those of the vertebral column, and it would appear that this weakness is greater in the muscles of the homolateral side. We have been able to observe this only in certain cases.

Paresis of the trunk muscles was most definite for variable periods after operation. These patients were unable to sit up in bed without support for any length of time, they could not straighten their backs and fell limp in any direction. This seemed to be due to the inability of the spinal muscles to bear the weight of the body. In cases which recovered a definite degree of lordosis when standing persisted when most of the other symptoms had disappeared.

In cases of extra-cerebellar tumour there was seldom such definite paresis of the homolateral limbs as was observed in the intra-cerebellar growths, except after operations from which additional cerebellar lesions resulted. On the other hand, we frequently found evidence of spastic paresis of the contralateral limbs and accompanying reflex changes characteristic of organic hemiplegia. This was undoubtedly due to the direct pressure of the tumour on the cortico-spinal system in the pons, and of this there was definite evidence in the fatal cases. This association of contralateral hemiparesis with cranial nerve palsies on the side of the lesion presents the clinical picture of crossed hemiplegia. In one or two advanced cases the pressure had been so great as to affect both pyramidal tracts.

Atonia.—Associated with the unilateral weakness there is often a defect of tone in the muscles of the homolateral limbs, so that during rest they assumed unwonted positions, and when handled are found to be limp and flaccid. The muscles may be soft and flabby to the touch. When such a limb is held at a proximal segment and shaken the distal segments flop about like the limb of a flail, apparently unrestricted by any active or passive tone of the muscles. The greater degrees of this hypotonicity are met with early in cases of acute onset, and especially after operations in which the cerebellum has been considerably damaged. In the latter cases, though chiefly homolateral, the opposite limbs may also be slightly hypotonic, but quickly recover.

The relation between this atonia due to cerebellar lesions and the state of the tendon reflexes is inconstant. While the hypotonia of tabes dorsalis and acute peripheral neuritis is always accompanied by loss of the tendon reflexes, the hypotonia of cerebellar lesions may be associated with definite increase of these reflexes, or the latter may be absent or diminished in various degrees.

For the first few days after operation when the deficiency in tone has been most marked, we have invariably found the tendon reflexes absent, but their rapid return did not correspond with any objective change in the tone of the muscles, and they even became exaggerated before any definite increase of tone occurred. This forces us to the conclusion that the state of the deep reflexes is not any measure of the muscle tone in such cases.

The irritability of the muscles to direct percussion was often more brisk on the homolateral than on the opposite side.

There is one phenomenon which we have repeatedly observed associated with, and apparently due to, hypotonia When a person attempts to execute a movement against resistance with a normal limb, and the resistance is suddenly removed, the limb rapidly moves a short distance in the desired direction till abruptly checked, or it may recoil. In a spastic limb the amount of recoil is excessive; in functional cases there may be neither primary movement nor recoil. In homolateral cerebellar disease the range of movement is excessive, generally continued till it is no longer mechanically possible, and there is little or no recoil. This absence of recoil must be attributed to the inability of the antagonistic muscles to react in tone to the sudden strain to which they are exposed (reflex muscle tone).

The simplest way to test this phenomenon is to request the patient to flex the forearm as strongly as he can, the elbow being firmly supported and the movement effectually resisted by the observer grasping the wrist. The resistance is suddenly relaxed by releasing the wrist.

We have rarely noted any considerable change in the tone of the muscles of the homolateral limbs in cases of extra-cerebellar tumour. With evidence of pressure on the pyramidal tracts there may be varying degrees of hypertonicity of the muscles, especially of those of the contralateral limbs.

Ataxia.—Some degree of ataxy of the homolateral limbs is one of the classical signs of unilateral cerebellar affections. The defect consists in a deficient control of the various component muscular contractions by which a complex act is executed, and in a disturbance of the normal association of the action of muscles with that of their antagonists, which regulate or limit the act. It is, in fact, a dysmetria. The individual components are ill-judged, being generally in excess of the requirement and inaccurately associated in time, the result being that the purposed act is only attained after several deviations from the direct line of movement. A special character of the ataxy is that it is directly due to this lack of accurate co-operation and association between the individual muscular contractions and not to impairment of muscular sense or of the sense of position. In other words, its origin is central, not peripheral. The patient is unable to check the deviations though fully conscious of his error. Unlike the ataxy of tabes dorsalis the deviations are not increased when the patient is deprived of the aid of vision. The ataxy appears only in active movement of the limb, does not increase towards the completion of the action, and, finally, the limb remains steady when its object is attained. In these characters it is the counterpart of the irregularity of movement styled intention-tremor, met with in disseminated sclerosis. Cerebellar ataxy is within wide limits more or less characteristic, and when the type is once recognised there should rarely be any difficulty in distinguishing it from other irregularities of movement. It is most evident in cases of acute onset or during acute exacerbations of the other symptoms, and especially after operative destruction of the cerebellum.

In cases of chronic course or where the lesion has become latent the ataxy is, as a rule, less definite. This is especially so when the condition has become complicated by hydrocephalus; then it may be less typical and approximate to the intention-tremor type characteristic of disseminated sclerosis. This seems to occur most frequently in tumours involving the vermis. In disease of one lateral lobe the inco-ordination is typically present only in the homolateral limbs, but not infrequently there is, in addition, some degree of ataxy in the opposite limbs, though always less in degree. When the tumour is in the vermis, or extends into both lateral lobes, the inco-ordination is bilateral, but greater in the limbs of the side on which the lateral lobe is more affected, or it may be more pronounced on one or other side as the effect of the tumour varies. The inco-ordination affects the arm more than the leg. The reeling gait would seem to suggest that it is greater in the lower extremities, yet, on examination, it is found that this is due in great measure to the irregular action of the trunk muscles. We have often noted when the patient stands erect that an unnatural degree of alternate contraction of the erectores spinæ of the two sides occurs as he attempts to maintain or attain his proper balance. Patients who are so unsteady on their feet that they can scarcely walk, present a comparatively slight degree of inco-ordination in carrying out movements as they lie in bed.

We have not been able to make out any constant difference between the ataxy which results from cerebellar disease, and that met with in extra-cerebellar tumours.

Some slight degree of inco-ordination generally persists for many months after the disease has been removed by operation, and may be one of the last signs to disappear.

We have carefully investigated in a number of our cases the sign described by Babinski as pathognomonic of cerebellar disease, to which he has given the name "diadococinesia." It consists in the inability of the patient to accurately perform rapid alternate movements with the homolateral limb, though the individual movements are easily possible. It is tested by requesting the patient to pronate and supinate the forearm in rapid succession; this can be quite naturally executed by the contralateral limb, but the homolateral arm is moved slowly, awkwardly and irregularly. This phenomenon seems to forcibly confirm our analysis of cerebellar ataxy—that it is a dysmetria or defective control of muscular action, with inaccurate combination of the component muscular contractions.

It seems to us that this inability to rapidly repeat movements is dependent upon the defective cooperation of the muscles and their antagonists, due in part, at least, to the diminution of the reflex muscle tone described, and to the consequent failure of the antagonists to control the primary movement owing to their lack of tone.

We are not, however, in agreement with Babinski that it is pathognomonic of cerebellar disease, nor have we found it constantly present in all cases of cerebellar lesions where we have looked for it. It seems to occur equally in cerebellar and extra-cerebellar tumours.

Attitude.—In many cases of unilateral cerebellar and extra-cerebellar tumours the head is held in a more or less characteristic attitude. When the patient sits or stands it is slightly flexed to the side of the lesion and rotated so that the chin is directed towards the opposite shoulder and the occiput approximated to the point of the homolateral shoulder. Though very characteristic, this attitude is neither constant nor pathognomonic. Occasionally in cerebellar growths the head is held in the reverse position, and this "cerebellar attitude" of the head is met with in cases of pontine, mid- and, more rarely, of fore- brain neoplasms. It cannot, therefore, of itself be used as a positive sign of cerebellar disease or as a definite aid in its localisation.

In some cases of tumour of the middle lobe there may be a tendency to retraction of the head; in others the head may be flexed and bent forward until the chin rests upon the chest, especially when the patients walk. We have also observed in a large proportion of the cases lordosis in the dorso-lumbar region when the patient assumes the erect attitude. Some patients, however, stand with their body rather inclined forwards. This seems to be most common in neoplasms of the vermis, probably in those cases where the disease is chiefly situated in its anterior part, and we have noted it specially after operation. Two of the patients when first placed upon their feet assumed a sprawling attitude, which was probably due in part to the general asthenia. We have not seen any tendency to pleurotonus or lateral curvature of the spinal column, though in unilateral disease patients are often inclined to bend over to the side of the lesion.

Involuntary Movements.—In unilateral cerebellar tumours which are not complicated by hydrocephalus, the homolateral arm is held remarkably steady when extended horizontally in front of the patient. The absence of any tremor, vibratory muscular contraction or spasm and the ability to maintain one position for considerable periods, is striking in a limb whose volitional movements are inco-ordinate, and emphasises the purely kinetic character of the ataxy. The contra-lateral arm may be unsteady or tremulous when held in the same position, but not as a rule to an unnatural degree.

In extra-cerebellar tumours, on the other hand, there is often irregular tremor in the horizontally extended arms, as a rule equally well marked in the limbs of either side. This tremulousness is in striking contrast to the unusual steadiness noticed in the homolateral arm in cases of unilateral cerebellar disease.

In a few cases there was for variable periods after operation some tremulousness of various parts of the body when unsupported, but as a rule it did not persist for long. It was most frequently observed in the head and trunk; when the patient sat up or was placed in the erect position there were constant nodding or bowing movements of the head and body, and at times lateral oscillations. There was similar spontaneous movement of the limbs, especially of the homolateral, when under analogous conditions. The muscular contractions from which they resulted seemed to occur only in the relaxed muscles, as the limbs remained fairly steady when rigidly extended in front of the patient. This condition seems to be identical with the astasia observed in animals after destructive lesions of the cerebellum.

Gait.—The gait of cerebellar disease has been accurately described as drunken, staggering, or reeling, but further analysis of its characteristics is necessary before it can be used as an aid in localisation of the lesion. There appear to be two distinct components which characterise the gait peculiar to unilateral cerebellar disease. (1) A tendency to stagger and stumble to the side of the lesion; (2) a tendency to deviate from the desired direction towards the side of the lesion. The stumbling is more or less characteristic; the patient when walking suddenly totters as though to save himself from falling, as if he were drawn by an unseen influence towards the side of the disease, but, as a rule, he quickly recovers his balance and direction. He may occasionally stumble to the opposite side, but he does so less frequently and to a less extent. This stumbling is rarely of such a kind as to suggest that it is due to weakness or giving way of the homolateral leg.

The deviation from the desired line of progression is not less characteristic, but often requires to be carefully analysed. In acute unilateral cerebellar lesions there is a constant tendency to deviate to the side of the lesion, so that, were it not corrected, the patient would describe a definite circus movement to this side, *i.e.*, with the homolateral shoulder towards the centre of the circle. But the patient is fully conscious of this tendency and almost invariably attempts to correct it, and when the symptoms have persisted for some time he may do so almost automatically. This he does by rotating the trunk so that the homolateral shoulder is held in front of, and generally higher than, its fellow, the transverse axis of the body being thus directed from the line of progression towards the side opposite to the lesion. Thus he substitutes for the potential angle of deviation towards the side of the lesion a virtual angle of deviation towards the opposite side, by means of a complementary rotation of his trunk in that direction through an equal angle.

When the disease has lasted for any considerable time, especially when the patient is daily up and about, there may even be an over-correction of the original deviation, so that he tends from the direct line towards the healthy side. This is seen on those days when the patient's gait is better than usual. If this over-correction is not fully recognised it may lead to misinterpretation and wrong localisation. Other patients may attempt to correct the tendency to deviation and stumbling by throwing the weight of their body as far as possible on the contralateral foot by leaning to that side, but this is of rare occurrence. The actual tendency to deviation from the direct line of progression towards the side of the lesion seems to be the essential condition. In acute cases it has always been very definite and was often recognised and spontaneously described by the patients as a sensation of compulsion or traction in that direction. For instance, a patient with left extra-cerebellar tumour, was walking very unsteadily when first examined, and spontaneously remarked, " I know I am always going to the left, but I can't help it; I feel as if something were pulling me over." Next day his gait was much better, and he deviated to the right, which he thus explained, " I am always inclined to go to the left and try to overcome it by going to the right, and sometimes when I am steadier I go too much to the right."

There is no constancy in the direction in which the patients, collectively or individually, turn round; as a rule they stop for a moment, and then turn slowly and awkwardly, more often perhaps round the side of the lesion.

In many cases, and, indeed, most commonly, the patient holds the homolateral leg more abducted than its fellow, and also rotated at the hip, so that the foot and toes point outwards. In this way he broadens his base. We have also noted frequently a definite ataxy or awkwardness of movement of the leg of the affected side as the patient walks, and he can always stand more steadily on the leg opposite to the side of lesion than on the homolateral. When the feet are placed together, the tendency is generally to fall backwards and to the side of the lesion.

In mesial tumours, or those involving both lateral lobes, the gait is more truly drunken or ataxic, and the patient stumbles and reels from side to side. In these cases there is often a tendency to fall backwards or forwards rather than to either side, and in one of our cases at least there was definite retropulsion; the patient when placed on his feet constantly stumbled backwards, as though pushed, at the same time deviating to the side most affected by the lesion, so that when supported he described a circle with the homolateral shoulder towards its centre.

In extra-cerebellar tumours which exert pressure on the pyramidal tracts in the pons the gait may be complicated by definite spastic paresis of the opposite leg, so that the patient may stand better on the homolateral leg than on the opposite one, and may stumble on the spastic limb.

In all cerebellar lesions the subjective uncertainty and unsteadiness seem to be often in excess of the objective disturbance. After operation the patient may be unable to sit up or walk for some time. When improvement sets in, he walks in the manner described above but with all the features more marked. Recovery is often rapid, but it may be some months before he can walk quite naturally. In several cases the lordosis and the stumbling to the side of the lesion were the last signs to disappear.

SENSORY SYMPTOMS.

Alteration of cutaneous sensibility never occurs in cases of cerebellar disease except when the tumour involves the pons, either directly or indirectly, by pressure, or causes secondary softening. Even in extensive lesions of the cerebellum, as where one lateral lobe has been practically destroyed, there has been no appreciable change in any form of sensation. The sense of position of the limbs, stereognosis, and the correct appreciation of weight, are also never impaired. Some anæsthesia over the distribution of the trigeminal nerve, however, may result from direct pressure on its sensory root, especially in cases of extra-cerebellar growths.

CRANIUM.

In the younger patients there may be definite bulging of the skull in the occipital region over the seat of the tumour. We have been unable to observe this in any of our adult cases, though some thinning of the skull was occasionally found on operating. In the majority of the cases there has been considerable tenderness on pressure over the occipital region, generally greater on the side of the disease. Auscultation and percussion of the skull never yielded positive signs.

MENTAL CONDITION.

Cerebellar growths do not seem to have any effect on the psychical functions, *per se*, though any acute increase of intracranial pressure may cause a drowsy, or stuporose state, as a rule of brief duration.

REFLEXES.

Tendon reflexes.—The variability of these reflexes is one of the most striking signs of cerebellar lesions. In some cases they are increased, while in others they are diminished or lost, but any one case may present both extremes within short periods. The knee and ankle jerks illustrate this fact best, not only because they vary more, but the degree of change is most noticeable in them. In many cases where the tendon reflexes of the upper extremities have remained natural or brisk throughout, those of the knee and ankle were at different times diminished or absent. In unilateral cerebellar tumours diminution on the side of the lesion is perhaps the most common condition, though an increase on both sides, or on the side of the lesion, is often met with. We have never obtained ankle clonus in such cases. In tumours involving the middle lobe there is generally an increase in the deep reflexes on both sides, and this condition is rarely absent when secondary hydrocephalus has developed.

In extra-cerebellar cases the tendon reflexes are rarely diminished; in some they remain natural, but as a rule they are increased, especially on the contralateral side. We have never observed any relative difference between the tendon reflexes of the upper and lower extremities. Ankle clonus is found fairly frequently associated with spasticity of the limbs opposite to the lesion, and in a few cases it may be present on both sides.

In all the cases which recovered we have noted absence of the tendon reflexes for some days following operation; they, however, quickly reappeared, and always on the contralateral side first. Their loss coincided with the extreme muscular asthenia and atonia to which we have already referred, but their reappearance and subsequent increase was not synchronous with any appreciable improvement in the muscular tone.

In some cases ankle clonus developed in the homolateral side while the asthenia and atonia of the muscles still persisted. This ankle clonus was scarcely identical with that met with in spastic states; it was irregular, intermittent, and of short duration, and was unaccompanied by any change from the flexor type of plantar reflex, or other evidence of interference with the pyramidal system. The presence of ankle clonus in these conditions is noteworthy, as attention has not yet been drawn to its occurrence.

Superficial reflexes.—These are usually natural in character, except in those cases in which there is pressure on the cortico-spinal system. When hydrocephalus has developed they may be diminished or absent on both sides, and when one pyramidal system has been pressed on they are diminished on the opposite side.

Plantar reflex.—We have never obtained an extensor plantar response in any case of uncomplicated intracerebellar lesion.

In extra-cerebellar tumours, on the other hand, where there has been pressure on the pons, an extensor plantar response may be found on the opposite or on both sides. Despite the large amount of compression these tumours exert on the pons a contralateral extensor response on plantar stimulation seems to occur only in the later stages, and in some cases has been absent throughout their whole clinical course.

FITS AND SPASMS.

In cerebellar cases a history of "fits" was occasionally obtained from the patients or their friends, but on closer questioning it generally appeared that these were attacks of vertigo of the severer form with clouding of consciousness.

We have, however, observed in one case a long series of seizures of peculiar nature.

In case 9, in which a large tumour lay in the under surface of the vermis, the operation by which its removal was attempted resulted in considerable hæmorrhage into the substance of the left cerebellar hemisphere, chiefly in the region of the dentate nucleus. This patient had a series of tonic spasms all more or less alike. The first occurred immediately after the operation while he still lay on the table recovering from the anæsthetic (chloroform). The face was unaffected by spasm, and respiration, which was slow and deep, was also undisturbed. Both arms were rigid and resistent to passive movement, the left more so than the right; they were adducted to the sides, extended at the elbows and wrists, while the fingers were flexed at all joints and covered the thumb. The left arm was rotated strongly inwards at the shoulder and the fore-arm pronated so that the ulnar border of the hand faced forwards, while the right arm was rotated slightly outwards and the fore-arm supinated till the palm of the hand came to look forwards. Both lower extremities were rigidly extended at hip, knee, and ankle, the rigidity being greater in the left. The left limb was rotated inwards and the foot inverted, the right rotated outwards with the foot everted. The position of the back was not fully observed in this fit, but there was a tendency to roll over to his right side. The spasm was purely tonic in all limbs, and uniformly maintained for about three minutes.

At the onset of the fit the eyes were in conjugate deviation to the right, but in its later stages they rolled slowly from side to side, their axes, however, not remaining parallel, as the eye of the side towards which the movement took place started first. There followed, immediately after its cessation, two similar fits in close succession.

The child never fully regained consciousness, though the corneal reflexes were present, and he occasionally cried out. After an interval of about an hour and a half the fits recurred.

As in the fit described, the onset was sudden without local commencement, and there were no clonic movements. The face was not affected, the head was retracted and rotated to the right, the back arched and laterally curved so as to be concave to the left, and the body rotated on the vertebral column to the right, so that it seemed as though he were rolling over to this side as he lay on his back. The position of the arms was as above described, except that in the severer fits there was flexion of the wrists. The rotation of the limbs, inwards at the left hip and shoulder, outwards at the corresponding joints of the opposite side, as well as the pronation of the left fore-arm and supination of the right, became more marked. When the spasm was strong, the adduction of the left limbs was pronounced and the right were abducted from the side.

The eyes behaved as in the first fit, that is, during the early stages both were in extreme deviation to the right, while later, as the spasm began to pass off, they rolled slowly from side to side, the eye of the side towards which the deviation was occurring beginning the movement and retaining its lead till it reached the outer canthus, though not to the full extent, as the other eye followed at a quicker rate. When both eyes had reached the limit of range to the one side, they commenced to move in the opposite direction in precisely the same manner. The movement of each eye was slow and uniform, a single excursion occupied about ten seconds.

The patient only survived the operation about nine hours, but during this time he had a very large number of seizures, which were carefully observed. They were all of the same type as those described, but the retraction of the head, the opisthotonus and the lateral curvature of the spine with the concavity to the left and its rotation to the right became more marked. In these later fits, adduction of the left limbs and abduction of the right were constantly observed.

Each fit passed off gradually, the tonic spasm slowly relaxing, first in the right limbs. The limbs were remarkably flaccid and atonic in the intervals between the seizures.

Summarising from these descriptions it will be seen that each fit consisted in rotation of the head, trunk and limbs on their longitudinal axes from the side of the lesion to the healthy side, and deviation of the eyes in the same direction. The homolateral limbs were also adducted, and the contralateral abducted. There were no clonic movements at any stage of the many seizures observed. Their onset was abrupt, but the relaxation of the spasm gradual. In many of the fits the tonic spasm was greatest in the muscles of the trunk, and in all of them it was more pronounced in the homolateral limbs than in the opposite.

We have also had the opportunity of observing a few seizures which affected the trunk and limbs in an exactly similar manner in the case of a girl where a large tumour infiltrated the pons and one lateral lobe of the cerebellum.

It is probable that these phenomena must be ascribed to an irritative lesion of the cerebellum. Those in the case we report were probably due to the recent hæmorrhage, and their short duration and frequent recurrence suggest that they were irritative phenomena. We have never observed similar seizures in forebrain lesions, and in the fact that they were purely tonic they seem to be the counterpart of the clonic spasms which result from cortical discharge.

Dr. Hughlings Jackson has described a variety of tonic cerebellar fits which occur in cases of tumours of the vermis. In these there is retraction of the head, arching of the back with flexion and supination of the elbows, rigid extension of the legs and pointing of the toes. We have never observed such, but it is conceivable that they are due to a compound of discharges similar to those we described from each lateral lobe of the cerebellum, dependent upon disease of the middle lobe.

A few of our other patients have given accounts of jerking movements of the homolateral arm, often associated with attacks of vertigo. In one case of extra-cerebellar tumour (No. 18) in which such involuntary movements were observed, they preceded the vertigo and consisted of an isolated jerk, or a short series of jerks, of sudden shock-like character of any part of the homolateral arm, lasting over a period of two to three minutes. The movements were irregular in distribution and sequence, and according to the patient occasionally affected the contralateral arm simultaneously, but always in slighter degree.

DIFFERENTIAL DIAGNOSIS.

It seems to us that when due attention is paid to these symptoms and physical signs the accurate diagnosis and localisation of the majority of uncomplicated cerebellar neoplasms is possible. On the one hand, it may be asserted, from the examination of the records of a great number of cases observed in past years in the National Hospital, that cerebellar tumours rarely, perhaps never, run a latent course or fail to produce clinical symptoms, and, on the other, this typical symptomatology is never met with in other conditions unassociated with other signs.

In considering the question of diagnosis, supratentorial tumours may, in the first instance, be placed in contradistinction to those situated in the posterior fossa and can, as a rule, be easily distinguished from them. Within the latter class are those seated in the substance of the brain-stem, those within the cerebellum, and those which lie without and only affect these parts by pressure. It is the differential diagnosis between these three varieties which we propose to discuss.

(a) *Between Cerebellar and Extra-cerebellar Tumours.*

As a rule the cardinal symptoms of intracranial neoplasm —headache, vomiting and optic neuritis—appear early and in pronounced degree in intra-cerebellar growths, while in cases of extra-cerebellar tumour any or all of them may first occur late in the history of the case. In the former class the functions of the sixth, seventh and eighth cranial nerves may be impaired, but always in slight degree. In extra-cerebellar tumours the affection of these nerves has been invariable and often considerable. The position of the growth in each case will naturally determine which of them will suffer most, but in those we have observed, marked or complete deafness resulted early, and there was generally definite affection of the facial nerve, which might even advance to complete palsy, with electrical reaction of degeneration.

Evidence of pressure on the motor or sensory roots of the trigeminus is rarely met with in cerebellar tumours, but commonly occurs, often in considerable degree, in the extra-cerebellar group. Weakness of the palate or vocal cords, with difficulty in swallowing and affection of phonation or articulation, seems to belong to the symptom-complex of extra-cerebellar tumours, as we have never met with these symptoms in uncomplicated cerebellar disease.

Marked affection of the functions of the cranial nerves, and especially disproportionate palsy of one, is, other things being equal, strongly in favour of the diagnosis of extra-cerebellar tumours.

Vertigo when present may be a useful aid in localisation. In both classes the movement of objects in front of the patient was always, in our cases, from the side of the lesion to the opposite side, but while in intra-cerebellar lesions the subjective rotation of self is in the same direction, in cases of extra-cerebellar tumours it has always been in the reverse.

In extra-cerebellar tumours the homolateral paresis and hypotonia is often absent or is less marked than in cerebellar lesions, but we have noticed no difference in the degree or quality of the ataxy or in the character of the gait. As above-mentioned, the hands are generally held remarkably steady in intra-cerebellar lesions, especially that of the homolateral side, but in extra-cerebellar we have frequently found considerable coarse tremor of the horizontally extended limbs, which, in some cases, became so accentuated on volitional movement as to resemble intention-tremor.

The association of cerebellar symptoms with crossed hemiplegia of even the slightest degree is strongly suggestive of an extra-cerebellar tumour compressing the pons. In our experience, an extensor plantar response absolutely excludes uncomplicated cerebellar disease.

Tumours situated in the middle lobe of the cerebellum can rarely produce such symptoms as to necessitate the serious consideration of their differentiation from lateral recess growths.

(*b*) *From intrapontine tumours.*—The symptomatology of intrapontine tumours varies immensely according to their situation and nature. In soft-infiltrating gliomata, such as form the pathological condition formerly known as "hypertrophy of the pons," the cranial nerves may be remarkably spared, while if the growth is firm and circumscribed it destroys all neighbouring structures indiscriminately, and produces quite a different clinical picture. It is possible to confuse the symptoms of the first variety with those of intra-cerebellar lesions, while those of the second variety tend to resemble those of extra-cerebellar tumours. The almost invariable absence of optic neuritis, the less intense headache and vomiting, and the early appearance of evidence of involvement of the cortico-spinal system in pontine tumours generally allows a correct diagnosis.

The inco-ordination which results from intrapontine lesions may resemble that seen in cerebellar tumours, but it is, as a rule, less definite in quality and situation. Cranial nerve symptoms, when present, are often bilateral and more general than is the rule, even in extra-cerebellar tumours, when one or two nerves involved in the growth are affected out of all proportion to the others. Such complete and permanent palsy of lateral conjugate deviation of the eyes as very frequently results from intrapontine growths, never never occurs in cerebellar or extracerebellar tumours.

Tumours situated further cerebralwards in the quadrigeminal or midbrain regions may produce some cerebellar symptoms, but the impairment of function of the cranial nerves of this level, with or without crossed hemi-paresis, and, perhaps, associated with hemi-tremor or hemi-anæsthesia, prevents, as a rule, any confusion.

The following table shows the chief points to be considered in the differential diagnosis of these three varieties of cases.

Symptoms and Signs.	Lateral Cerebellar Tumours.	Extra Cerebellar Tumours.	Intrapontine Tumours.
Optic neuritis ...	Early and intense ...	Variable	Often absent or late.
Vertigo	Subjective rotation of self from the side of the lesion	Subjective rotation of self to the side of the lesion	Indefinite.
Cranial nerves V.	Rarely affected	Often affected... ...	Affection of these nerves often bilateral. Paresis may be supranuclear or nuclear, and grouped according to nuclear arrangement. Paralysis of a nerve on one side and of an adjacent or distant nerve on the opposite side. Permanent paralysis of conjugate deviation of the eyes.
,, ,, VI.	Weakness of conjugate deviation to side of lesion. Weakness of external rectus on side of lesion. Slow deliberate nystagmus to side of lesion	Same as in unilateral cerebellar tumours	
,, ,, VII.	Paresis slight if present	Paresis more marked	
,, ,, VIII.	Deafness on side of lesion incomplete and variable. Tinnitus general	Deafness on side of lesion marked—generally complete. Tinnitus referred to ear on side of lesion	
,, ,, IX.	Never affected	Occasional paresis on side of lesion	
,, ,, X.	Ditto	Ditto	
,, ,, XI.	Ditto	Ditto	
,, ,, XII.	Ditto	Supranuclear paresis on contralateral side	
Motor system ...	Homolateral paresis, ataxia and atonia	Homolateral paresis and ataxia; contralateral spastic paresis common—occasionally bilateral	Paresis often bilateral, with spasticity. Ataxia general.
Sensory system ...	No change	No change	Occasionally hemianæsthesia.
Reflexes—Tendon	Variable, often diminished	Generally increased, especially on contralateral side	Increased, often unequally.
,, Superficial	Normal	Often diminished on contralateral side	Diminished, often unequally.
,, Plantar	Flexor	Flexor or extensor. Extensor on contralateral or both sides	Extensor on one or both sides.
Sphincters ...	Not affected	Rarely affected ...	Generally affected.

THE RELATION OF THE SYMPTOMATOLOGY OF CEREBELLAR DISEASE IN MAN TO THE RESULTS OF EXPERIMENTS ON ANIMALS.

It is important to consider how far the symptoms of cerebellar tumours are directly due to destructive cerebellar lesions, and to what extent the cerebellar symptoms may be hidden or replaced by irritative or paralytic phenomena according to the extent and degree of pressure the tumour asserts on other parts. This is the more important as many physiologists refuse to accept clinical evidence in discussion of the functions of the cerebellum, owing to the possibility of complication of cerebellar symptoms by indirect or distant lesions.

It seems to us that this is partly unjustifiable. The effect of the increase of intracranial pressure is the first disturbing factor to be considered, but even with infratentorial growths it is fairly uniformly distributed over the whole intracranial cavity and is not liable, except when of long duration, to produce focal disturbance, and then most severely affects the forebrain by dilatation of the ventricles. Its clinical manifestations are well recognised and known to rarely produce such focal symptoms as can be confused with cerebellar disease.

That cerebellar tumours may produce accessory symptoms by direct pressure on the neighbouring structures, especially on the cranial nerves and brain-stem, is evident, but these indirect effects can be easily excluded, and from a knowledge of the symptomatology of lesions of these parts, the true cerebellar signs can be recognised by exclusion. The accumulation of a large amount of clinical observation makes this possible.

Finally, from observation of the effects of destructive lesions entailed by operation (and in several of the cases which succumbed there was destruction of practically the whole of a lateral lobe as complete as is possible by experiment) it was evident that their symptoms were identical in character with those of cerebellar tumour.

It is for various reasons very difficult to compare the symptoms of cerebellar disease in man with the results obtained by experimentation in the lower animals.

In the first place there is, according to all who have studied the subject, a close relation between the functions of the cerebellum and cerebrum, and the latter is able to compensate, to some degree, for any loss of the former. Thus, while the symptoms of ablation of one cerebellar hemisphere of a dog soon subside under normal conditions, practically no recovery takes place, as Luciani has shown, after extirpation of the contralateral sensorimotor cerebral cortex. The permanent symptoms of a cerebellar lesion must consequently depend in part on the possible extent of this compensation, and as the relative development of the forebrain is greater in man, the power of compensation should, *a priori*, be greater. As illustrative of this may be cited the observations of Luciani and Russell that the symptoms which follow partial lesions of the cerebellum are less intense in the ape than in the dog, and that in the former the compensatory process sets in earlier.

Secondly, the divergence between the results of experimental investigators and the still greater diversity of their interpretations, make it impossible to form a final conclusion on the symptomatology of ablation or stimulation of the various portions of the cerebellum.

Thirdly, the erect attitude of man, the change in the plane of locomotion, and the narrowing of the biped basis, influence the disturbances of locomotion which result from cerebellar disease to such an extent that comparison with the lower mammals is difficult.

And finally, the influence of lesions depends not only on the extent of the destruction they cause, but equally on their rate of onset. In cases of cerebellar tumour of relatively slow growth, there is considerable time and opportunity for compensation by intact parts of the organ (organic) or by other parts of the nervous system (functional). As we, however, possess abundant observations on cases for variable periods after operation, from which large acute lesions resulted, we shall rely mainly on the facts culled from them for comparison with the experimental results.

Luciani, Russell and Thomas record paresis of the homolateral limbs after ablation of one cerebellar hemisphere, but Ferrier, and Ferrier and Turner were never able to observe it. In several cases, however, there was definite weakness of these limbs when compared with their fellows and it was invariably pronounced after operation, though never of long duration in any great degree. Besides this unilateral paresis, many cases in which the lesions were acute were for some time in an extremely asthenic state, and had little command of muscular power. We are in agreement with Russell that the weakness is as a rule more marked in the upper than in the lower limb, though Luciani describes the leg as the more affected. In several cases of unilateral lesions we have carefully examined the conditions of the spinal muscles, but were rarely able to observe any relative weakness upon either side. In extensive or acute lesions, however, the patient may be unable to hold his head or trunk erect, and this bears out the undue weakness of the trunk muscles which has been observed in animals with extensive cerebellar destruction. As it is possible to exclude affection of the cortico-spinal system as the cause of the weakness, it seems that the available evidence must be accepted as conclusive that homolateral paresis may result directly from cerebellar disease. In man it is easy to make the subject exert his full power and accurately estimate and compare that of the two sides; this is not possible in animals.

In several cases, and especially after operation and in those with acute onset, we found marked hypotonia of the muscles of the homolateral limbs, as Luciani has described. We have, however, never observed any tonic spasm or rigidity in these limbs in the earlier stages, as described by Luciani, Russell and Thomas. On the latter point our observations are in agreement with those of Ferrier, though he did not find hypotonia during any stage after the ablation. The state of the tendon reflexes cannot be regarded as a measure of the tone of the muscles, at least in cerebellar disease. The flaccidity and flail-like character of the limbs when handled, the lack of the normal resistance to passive movement, and the soft and flabby consistence of the muscles to touch, are the indications on which our statements are based.

Astasia, or constant fine tremors with wider oscillation on volitional exertion, is, according to Luciani, the third cardinal symptom of cerebellar lesion, and has also been described by Ferrier and Turner, Russell and Thomas. This has not been a symptom of any of our uncomplicated cases of cerebellar tumour, but in a few cases there was considerable tremor of this variety of the head, trunk, and of the limbs, greater in those of the same side for some days after operation if the parts were unsupported. The unsteadiness only persisted for long in two cases, and was finally limited to the head. An accentuating irregularity of movement of the intention tremor type was then also present, but this was rarely observed in uncomplicated cases of tumour. This condition of tremulousness which follows operation is remarkable and contrasts with the almost unnatural steadiness of the homolateral arm when it is horizontally extended in front of the patient, as described by Babinski, and confirmed by our observations. Reference may here be made to the class of tremor occasionally met with as a clinical symptom, which has been attributed by one of us to lesions of the cerebello-rubral system, though it has not appeared in any of the cases we describe.

Homolateral inco-ordination has been almost universally recognised by experimental investigation as a symptom of cerebellar lesions. Luciani, however, refuses to regard it as an essential symptom of cerebellar disease; according to him, it is due to a combination of the paralytic and compensatory phenomena, while the purposeless and ungraduated actions of the limbs which he styles dysmetria, should result from the asthenia and atonia of the muscles. The facts we have observed do not allow us to agree with these views. True cerebellar ataxy is often demonstrable in chronic cerebellar lesions where there must be a state of equilibrium between the paralytic and compensatory symptoms, and it may be unassociated with asthenia or atonia, though if it is, as we consider it, a primary and essential symptom of destructive lesions of the cerebellum, it will, as a rule, coincide in degree with these.

In man ataxia, atonia and paresis appear to be, and in this order, the essential signs of cerebellar disease.

We have seldom observed in man the pleurotonus, with concavity to the side of the lesion, which most experimenters save Ferrier and Turner have described, though there was often visible, when standing, a forced inclination of the trunk to that side. The position of the head in patients with unilateral cerebellar tumours often corresponds to that in animals after removal of the one cerebellar hemisphere, *i.e.*, the chin is directed to the opposite side, and the head is slightly inclined to the lesion. Neither retraction nor flexion of the head was a marked symptom in any of the cases here considered, but we have observed a pronounced tendency to retraction or falling back of the head when the patient sat up in two cases in which disease of the vermis was diagnosed but not confirmed. As in animals with unilateral cerebellar loss, there is often in man a tendency to abduction of the homolateral leg in walking.

Rotation movements were not a pronounced symptom in any of our cases, but it is necessary to mention the observations of the classical authorities when referring to the subjective and often objective rotation of vertigo and when discussing the significance of the series of rotation fits we describe. Unhappily, so much confusion has arisen from the terminology applied that we find it necessary to translate the descriptions of the forced movements into our own words. By rotation to the right we mean the movement which results if a man while standing erect turns on his vertical axis so that the left shoulder first moves forwards, or, if lying on his back, the left shoulder first rises so that he turns over to his right side.

Besides the confusion from verbal description, there is considerable disagreement among observers on this point. After ablation of one cerebellar lobe Luciani, Ferrier, Turner, Thomas and Muskens would describe the movement in our words as rotation from the side of the lesion, while Russell, Orestano and Pagano, as rotation to the side of the lesion. This forced rotation was only observed for a short time after recovery from the anæsthetic, and was not constant. The lesion which apparently most frequently results in rotation is simple section of the one middle peduncle, and then, according to Majendie, Ferrier and Curshman, the movement is in the same direction as after ablation of one cerebellar hemisphere, but Schiff and Luciani observed that the animals first rotated towards and later constantly from the side of the lesion, while Hitzig describes the rotation as towards the lesion. More recently Sergi, a pupil of Luciani, has drawn attention to the fact that the direction of rotation is not constant, and may occur in the one or the other direction. As a rule, when one hemisphere is affected, the animal rotates from the side of the lesion, while, if the peduncles are divided, the rotation occurs in the reverse direction. These facts may explain the discrepancies of the various observers, *i.e.*, whether the lesion be of the mass of the cerebellar lobe or of the peduncles, which are largely composed of afferent fibres to the contralateral hemisphere. This is of considerable importance in the consideration of our cases, as in extra-cerebellar tumours the middle peduncle is presumably the part which suffers most.

Russell has found that both section and irritation of the vestibular nerve causes rotation movement in the opposite direction to that which he observed after the removal of one lateral cerebellar lobe.

The most definite rotation-movements we have observed were in the case of cerebellar fits reported, and they were from the side of the lesion towards the healthy side. But in several cases there was a great tendency to lie on or roll over to the healthy side, especially after operation on the one cerebellar hemisphere. These patients would complain of feeling forced to roll over to this side apart entirely from definite subjective vertigo. In states of acute vertigo, on the other hand, where extra-cerebellar tumours compressed the auditory nerve or middle peduncle, the tendency has been to rotate on the vertical axis, as well as to fall towards the side of the disease.

The direction of the subjective rotation in attacks of vertigo, which so frequently occur in both cerebellar and extra-cerebellar tumours, is especially interesting in this connection. In the former class of case it was always from, in the latter towards, the side of the lesion. This acute vertigo must be regarded as an irritative phenomenon. As the rotation-movements in animals are, from their transient nature and their acute and periodic occurrence, probably of the same nature, our facts are in agreement with the majority of experimentalists excepting Russell as regards intra-cerebellar disease, while our clinical observations agree with his, that lesions of the auditory nerve produce the rotation (in our cases subjective) in the opposite direction to that which occurs in pure cerebellar lesions. It is difficult to understand this inverse relation unless it be due to the fact that each vestibular nerve and middle peduncle has chiefly a crossed connection with the cerebellum.

The apparent movement of external objects has been in both classes of cases from the side of the lesion. This is probably not dependent on a relative displacement of the visual fields secondary to the eye movements, as Mach believes, but is a reflex sensory phenomenon analogous to the sense of rotation of self.

Abnormal positions of the eyes have been observed by most investigators as a more or less persistent symptom after unilateral cerebellar destruction. All are agreed that the eyes are deviated to the opposite side, and in addition, there may be for short periods some irregular position. Luciani and Ferrier describe the typical skew deviation of Majendie, *i.e.*, the homolateral eye is directed downwards and inwards, the contralateral outwards and slightly upwards; but Russell observed the homolateral eye up and out, the other down and out.

We describe the same tendency to conjugate lateral deviation of the eyes to the healthy side in unilateral cerebellar disease, and have in addition repeatedly observed the Majendie "skew-deviation" for a few hours after operation, though in two cases this skew-deviation persisted for longer periods, in one case after operation, in the other apart from operative interference.

Clinical observations agree with Russell's assertion that nystagmus is most marked on looking to the side of the lesion.

It is difficult to compare the biped gait with that of quadrupeds, but as do most observers, we find in man a tendency to fall to the side of the lesion. This does not appear to be entirely due, as Luciani assumes, to paresis of the homolateral limbs, for we have failed to observe any relation between the tendency to fall and such paresis, or that this tendency occurs at the moment the patient throws his weight on the homolateral foot. To us it seems to be rather due to a deficiency in the proper co-operation of the muscles of equilibium, especially in those of the trunk and perhaps in part to paresis of these. The patient does not fall through any giving way of the limb, but as we have frequently observed in acuter cases, the body is definitely inclined towards the affected side till its centre of gravity approaches or exceeds the limit of its base. But the stumbling to the faulty side, as we have observed it, does not depend alone on this factor; there seems to be in addition an inequality in the muscular functions of the two sides, in favour of the contralateral, which results in a disturbance of the equilibrium.

We have not observed in any case of cerebellar disease such sensory loss as has been described by Russell, and one fact deserves special emphasis in view of the assertions of Lewandowsky and others to the contrary, that not even in cases of extensive lesions (*e.g.*, those following operations), was any impairment of the sense of position or of active or passive movements of the limbs to be made out under the most favourable conditions of examination. In man alone can sensibility be accurately tested, and here all evidence conclusively negatives any sensory function of the cerebellum.

The clonic irregularly distributed spasms of the homolateral limbs which we observed or received descriptions of in some cases, are probably analogous to those obtained by Ferrier and Russell (chiefly in the upper extremity), on electrical excitation of one lateral lobe of the cerebellum, and by Pagano on stimulation with curare.

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DESCRIPTION OF PLATES.

Figures 1, 2 and 3 are photographs of the brain of case 20 to represent the position of extra-cerebellar tumours and their relations to the neighbouring parts.

The sections represented in figs. 2 and 3 are not quite vertical to the plane of the brain-stem, but oblique from above, downwards and forwards consequently the degree of compression of the pons relative to that of the cerebellum appears greater than is actually the case.

Fig. 1 shows the position of the tumour on the base of the brain and the compression of the pons, and of the right lateral lobe of the cerebellum by it.

Fig. 2 is a section through the cerebellum and medulla oblongata at the level B, B, fig. 1. Seen from in front. It illustrates the compression of the right lateral lobe of the cerebellum by the tumour.

Fig. 3 is a section through the brain at the level represented by the line A, A in fig. 1. Seen from behind. It illustrates the compression of the middle cerebellar peduncle, and of the right lateral lobe of the cerebellum, and the backward displacement of the latter.

Fig. 4 is a photograph of case 7 taken a few weeks after removal of a tumour from the left lateral lobe of the cerebellum, to illustrate skew-deviation of the eyes.

PLATE I.

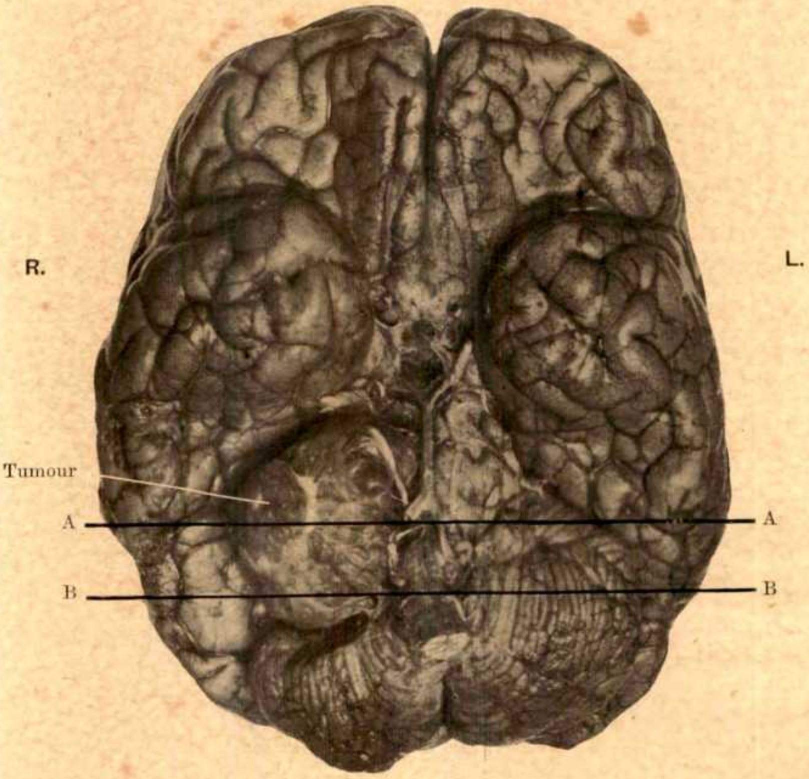

FIG. 1.

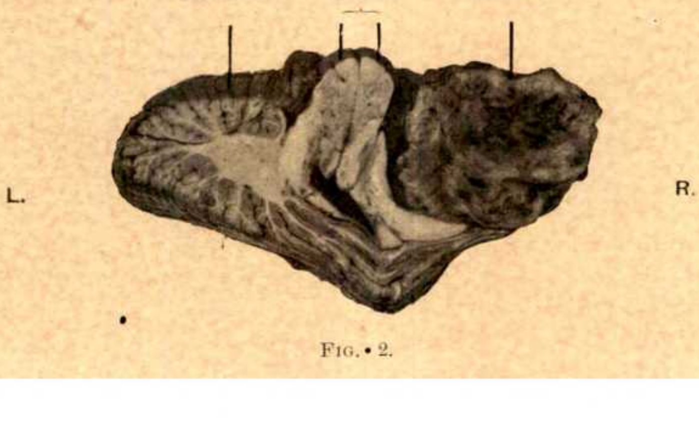

FIG. 2.

PLATE II.

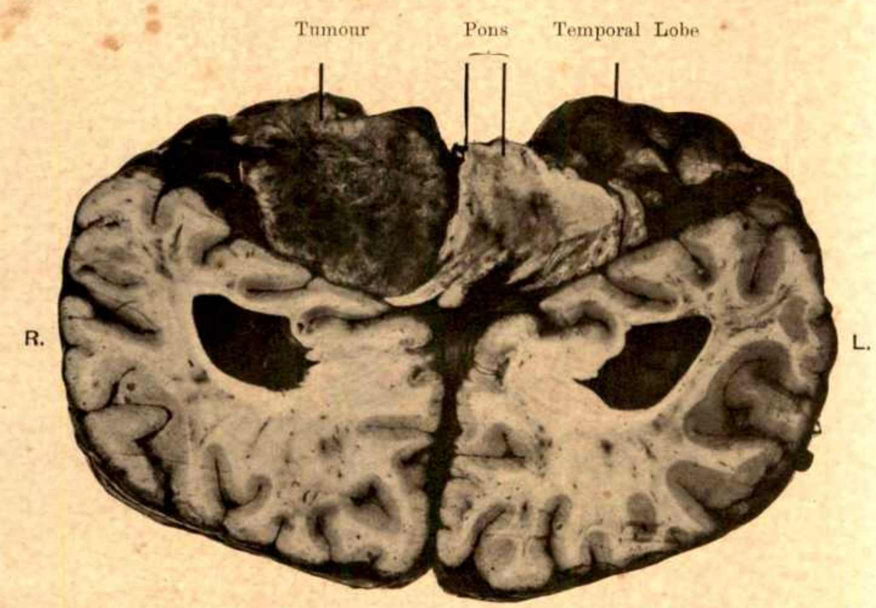

FIG. 3.

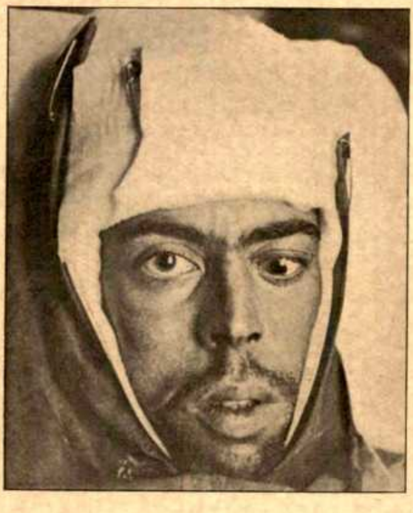

FIG. 4.

APPENDIX.

Reports of 22 Cases of Cerebellar and Extra-cerebellar Tumour which have been personally observed, and in which the Diagnosis has been confirmed by Operation or by Autopsy.

Case 1.—*Tubercular tumour of the right lateral lobe of the cerebellum.*

R. B., aged 24, female. Under the care of Sir William Gowers, 1904.

Family history and previous health unimportant.

For seven months headache, chiefly in the occipital region, and from there pain often radiated down her neck; frequently accompanied by attacks of vomiting. For five months had walked like a drunken person, staggered and fell, generally to the R. For four months her hands had been weak and unsteady, and she had held her head stiffly over to the R., and complained of diplopia, especially on looking to the R.

Had several fainting attacks in which she became cyanosed and fell backwards, generally associated with vomiting but never accompanied by convulsions.

Special senses.—Smell and taste normal. Hearing equally good on both sides, never tinnitus or vertigo. Vision very poor. Intense optic neuritis passing into atrophy.

C. N., 3, 4, 6.—Constant slight deviation of both eyes to the L. Slight weakness of R. external rectus.

Nystagmus on lateral movement, slow and forcible on looking to R., finer and more rapid on looking to L. It was not constantly present.

Pupils equal, reactions good.

7.—Slight weakness of the R. side of face on both expressional and volitional movement.

5, 9, 10, 11, 12.—Functions, normal.

Attitude.—Head generally retracted with occiput approximated to the R. shoulder. No other peculiarity.

M. S.—Muscles soft and flabby, especially those of the R. limbs which were definitely hypotonic. Strength of both limbs poor, R. considerably weaker than L. Co-ordination of finer movements impaired on both sides, considerably more on the R. than on the L. Pronation-supination test poorly executed with R. arm. No tremor or spontaneous movements.

Gait.—The R. shoulder was generally held higher than, and in front of, the L. No lordosis. The R. leg was abducted and rotated outward so that she stood on an unnaturally broad base. She could stand better on the L. leg than on the R. She tended to deviate to the R. of the line of progression and stumbled and fell to the same side. Less frequently the L. shoulder was held in front of the R., and she staggered and stumbled to the L.

Sensory system.—No defect in any form of sensation.

Reflexes.—Deep: Variable, the L. however being constantly brisker than the R., which were occasionally absent. Superficial reflexes equally brisk. Plantars flexor.

Cranium.—No bulging or local tenderness. No bruit audible on auscultation.

Ten days after admission the tumour was removed by Sir Victor Horsley. It was an almost spherical mass measuring about 3½ cms. in diameter, and lay in the R. lateral lobe of the cerebellum. The patient recovered rapidly from the operation.

For some days after, the phenomena above described were exaggerated, especially the weakness, hypotonia and ataxy of the R. limbs, and the tendon-reflexes were absent. From this time the patient gradually improved and there was considerable return of vision. After some weeks she was able to walk fairly well, though the gait was still reeling with a constant tendency to deviate and fall to the R. The constant conjugate deviation of the eyes to the L. was more marked and there was very slow, coarse nystagmus on lateral deviation to the R. The superficial reflexes remained normal; the deep were for a time absent on the R. and diminished on the L., but afterwards became brisk, though always more so on the L. than on the R.

Two months after the operation the patient developed tubercular meningitis and died. At the autopsy it was found that the greater part of the R. lateral lobe of the cerebellum had been destroyed.

Case 2.—Tumour (glioma) of the left lateral lobe of the cerebellum.

A. N., aged 27, female. Under the care of Dr. Ferrier. 1904.

The patient's family history was good, but she herself suffered from pulmonary tuberculosis and from disease of the R. middle ear. Three months before admission to the hospital she had severe pain in the back of the head and neck, associated with attacks of vomiting and "rushing noises" in the back of the head. Shortly after the onset of these symptoms diplopia developed. A month later her eyesight began to fail, and her hearing became more impaired. Three weeks before admission her walking became very unsteady, and she had attacks of giddiness, in which external objects seemed to move from L. to R. in front of her.

State on Admission.

A pale, delicate woman, with tubercular affection of both apices pulmonum.

Special senses.—Smell and taste normal. Hearing.—Bone conduction was much better on the R. side than on the L., though, owing to the condition of the middle ear on the R. side, aerial conduction was better on the L. than on the R. Vision.—R., $\frac{6}{18}$; L., $\frac{6}{18}$. Intense optic neuritis.

C. N., 3, 4, 6.—Weakness of L. external rectus. Slow and deliberate nystagmus on lateral movement in both directions, more marked to L. On downward movement the nystagmus was vertical, while on upward movement it was vertical, with a rotation to the L. Pupils were dilated, equal and reacted normally.

7.—Slight weakness of the L. side of the face.

5, 9, 10, 11, 12.—Functions normal.

Attitude.—Showed no peculiarity.

M. S.—Muscles were of equal size and natural in tone. The strength of the limbs was equal on the two sides. Co-ordination was definitely impaired in the L. limbs. Babinski's cerebellar sign was not definite. There was no tremor when at rest.

Gait.—She deviated to the R. but staggered to the L. She could stand better on the R. than on the L., and when her feet were placed together she tended to fall backwards and to the L.

Sensory S.—No defect.

Reflexes.—Deep; were brisk and equal on both sides. The superficial reflexes were normal. The plantar reflexes were flexor, and there was no sphincter affection.

Cranium.—There was no deformity or bulging, but the occipital region was tender on pressure, especially on the L. side. Mr. Ballance operated, and removed a large tumour from the left lateral lobe of the cerebellum in its posterio-inferior angle, just above the fourth ventricle.

The patient collapsed and died the same evening.

Case 3.—*Tumour* (*tubercular*) *of the left lateral lobe of the cerebellum.*

H. B., aged 18, male. Under the care of Dr. Buzzard. 1904.

This patient's mother had had syphilis, and he had tubercular disease of the L. hip and the R. wrist.

A year ago patient suffered from several severe attacks of occipital pain, which radiated down his neck. This, however, gradually passed off, and he felt well till five weeks ago, when the headache returned, accompanied by vomiting and vertigo. At these times he felt as if he were being twisted round L. to R., and external objects appeared to move in the same direction. He also had double vision on looking to the L., and noticed that he was becoming deaf in the L. ear. About a week later his walking became affected, and he staggered and tended to run into people on his L. side. On several occasions he complained of twitching of the L. side of his face.

State on Admission.

There was latent tubercular disease of both the R. wrist and L. hip, and the corresponding muscles were much wasted.

Special senses.—Smell and taste normal. Hearing.—Slight deafness in L. ear. Vision.—R., $\frac{6}{12}$; L., $\frac{6}{9}$. Well marked optic neuritis.

C. N., 3, 4, 6.—Weakness of both external recti, greater of the L. Nystagmus, slow, coarse and deliberate on looking to the L., fine and rapid on looking to the R. Vertical irregular nystagmus was also present on upward movement. The pupils were equal and reacted well.

5.—Slight wasting of the L. masseter, with deviation of jaw to L.

7, 9, 10, 12.—Functions normal.

Attitude.—The occiput was constantly approximated to the R. shoulder, the chin pointing to the L.

M. S.—Owing to the joint conditions the relative strength of the muscles could not be estimated. Tone of L. limbs subnormal. Co-ordination impaired in both upper extremities, and slightly in the lower, the ataxy being greater in the L. limbs. Babinski's cerebellar sign was not definite. Hands held very steady, especially L., when horizontally extended in front of him.

Gait.—Was reeling and unsteady, and he deviated and stumbled more to the L. than to the R.

Reflexes.—Deep; all diminished on the R., normal on the L.

Superficial; were brisk and equal; planta no sphincter trouble.

Sensory S.—Unaffected.

Cranium.—No bulging or tenderness.

The patient remained in much the sam observation, except for an increase of the

He was operated on by Sir Victor Ho being completed on May 3, 1904, when irregular shape, which measured about fou was removed from the L. lateral lobe of th to the surface on the dorsal aspect. operation well.

May 6, 1904.—Symptoms and sign except for definite weakness of the L. side

May 22, 1904.—All signs less marked, gait nearly natural. The deep reflexes b plantar responses flexor.

Six weeks after operation most of t were still present, but there was in add lateral deviation of the eyes to the L., and on attempting it. The facial and massete disappeared. There was slightly greate inco-ordination of the L. limbs. Gait as don reflexes diminished on L. Gait beca except for the complications due to the tul L. hip.

Case 4.—Cyst in left lateral lobe of the

S. C., aged 7, male. Under the care o

Patient's previous health and family hi

Three weeks previous to his admission headache and vomiting, and a few day dimness of sight. His condition rapidly developed a squint with diplopia, and sta walked. He also complained of transien The headache was chiefly in the L. fr regions.

State on Admission

Patient very dull and drowsy. Viscera

Special senses.—Smell and taste norma on L. Vision, only light perception. Int

C. N., 3, 4, 6.—Eyes deviated to the

external rectus, other ocular movements good. Slow, deliberate and coarse nystagmus on looking to L., fine and rapid on looking to R.

7.—Slight weakness of L. lower face.

12.—Tongue protruded slightly to the L.

5, 9, 10, 11.—Functions normal.

Attitude.—In bed he lay on his L. side, with the occiput approximated to the L. shoulder.

M. S.—The muscles of good size. The L. limbs much weaker and more flaccid than the R. Co-ordination much impaired in both L. limbs and to a less extent in the R. upper extremity.

Gait.—There was well-marked lordosis when he stood. He walked upon a wide base, the L. leg being somewhat abducted and rotated outward. He tended to drag the R. foot. Very ataxic, staggered to either side, and tended to fall to the right.

Sensory S.—Unaffected.

Reflexes.—Deep; brisk both sides. Superficial; abdominal diminished on the R., brisk on the L. Plantars flexor.

Cranium.—Slight bulging in L. occipital region, with tenderness on pressure.

A large cyst in the L. lateral lobe was opened and drained by Sir Victor Horsley. A very rapid recovery took place, and patient could walk a fortnight later. A month after operation his vision was $\frac{6}{6}$ in both eyes; the discs were muddy, but not swollen. There was slight lordosis and ataxy of the L. side. Slow nystagmus on looking to the L. The power of the L. side was much improved.

Six months after operation the only abnormal sign was a slight degree of lordosis. Reflexes normal.

Case 5.—Cyst of the left lateral lobe of the cerebellum.

A. W., aged 15, male. Under the care of Dr. Tooth. 1903.

The previous history unimportant.

Four months before admission patient suffered constantly from severe headache in the occipital region and had frequent attacks of vomiting. These symptoms were soon followed by dimness of vision and periods of transient blindness. His walking became affected a few days later, he staggered like a drunken man. Three months later he began to suffer from attacks of giddiness, in which he had no sense of rotation either of himself or his surroundings, but merely a feeling as if he must fall. During the last month he noticed unsteadiness of the L. arm, and in a less degree of the R. arm.

State on Admission.

Special senses.—Smell, taste, and hearing normal. Vision, R., $\frac{6}{36}$; L., $\frac{6}{18}$. Intense optic neuritis.

C. N., 3, 4, 6.—Slight weakness of L. external rectus. Ocular movements good. Nystagmus; coarse and deliberate on lateral movement to the L., more rapid on looking to the R. Pupils large, equal, reacted well.

5, 7, 9, 10, 12.—Functions normal.

Attitude.—Head generally rotated to the R., with the occiput approximated to the L. shoulder.

M. S.—Muscles were well developed and neither rigid nor flaccid. The strength of the L. limbs was considerably less than that of the R. Co-ordination was much impaired in the L. limbs, and slightly affected in the R. arm. Babinski's cerebellar sign was present in the L. arm. There was no tremor when at rest.

Gait.—The body was held slightly rotated to the R., the L. shoulder being in front of the R. There was slight lordosis. He walked in a staggering and drunken manner, but did not deviate to either side.

Sensory S.—Normal.

Reflexes.—Deep; all brisk and equal. Superficial; normal. Plantars flexor. No sphincter trouble.

Cranium.—No bulging, deformity, or tenderness.

Sir Victor Horsley removed a large cyst which lay deeply situated in the L. lateral lobe. It did not appear to be connected with any growth. The patient recovered well from the operation.

Headache and vomiting at once ceased, and other symptoms were relieved. For some days there was some weakness of conjugate movement of eyes to L., and very marked nystagmus on movement, especially to L.; also spontaneous nystagmus, with jerking to L. There was considerable paresis, atonia and ataxia of the L. limbs, but these symptoms soon disappeared. The L. tendon-reflexes were for a time diminished. Three weeks after operation he was able to walk alone, though in a staggering manner, and with a great tendency to stumble and deviate to the L.

Six months after operation all symptoms had disappeared, except some lordosis and a tendency to occasionally stumble to L. Later, his gait was quite normal.

Case 6.—*Cyst in the left lateral lobe of the cerebellum.*

E. B., aged 14, female. Under the care of Dr. Ferrier, 1904. The patient's previous health and family history, good.

The present illness came on three and a half months ago with headache and vomiting. The headache was bilateral and both occipital and frontal in situation; at first occasional, but latterly almost constant. Also giddiness, external objects appearing to go round in front of her though not in any particular direction. Her vision became weak about a month ago and rapidly deteriorated, and about the same time it was noticed her head was held stiff and was bent over towards the L. Diplopia also developed about this time. Fourteen days ago her gait became affected, she staggered and tended to walk to the L. For the last seven days she has complained of noises in both ears.

State on Admission.

Special senses.—Smell, taste and hearing, normal. Vision.—R., $\frac{6}{18}$, L., only light perception. Intense optic neuritis.

C. N., 3, 4, 6.—Ocular movements good, except slight weakness of L. external rectus; occasionally coarse nystagmus on looking to L. Pupils equal and reacted well.

7.—Slight weakness of the L. side of the face.

5, 9, 10, 11, 12.—Functions normal.

Attitude.—Occiput approximated to L. shoulder and chin pointed to R. Lay on R. side. Lumbar lordosis when erect.

M. S.—Development and tone of muscles, good. Considerable weakness of L. limbs, which were also ataxic, though the co-ordination of R. was good. Executed rapidly repeated movements equally well with both arms. No tremor or unsteadiness of hands.

Gait.—Rotation of vertebral column to R., *i.e.*, L. shoulder held in front of R. Could walk fairly well, but deviated and stumbled to L. Stood more steadily on R. foot than on L.

S. S.—Unaffected.

Reflexes.—Deep: Arm-jerks diminished and equal. Knee-jerks—R. diminished, L. absent. Both ankle-jerks diminished. The superficial reflexes brisk and equal and the plantars flexor. No sphincter affection.

Cranium.—Slight prominence in the L. occipital region, with great tenderness on pressure.

A few days after admission Sir Victor Horsley evacuated a cyst in the L. lateral lobe, which contained some clear straw-coloured fluid. The patient bore the operation well. A fortnight later the optic neuritis was rapidly subsiding with improvement of vision. The eyes were constantly deviated to the R. Weakness of conjugate deviation to L. Slow, deliberate nystagmus on looking to L. Head in position described above; limbs flaccid, especially the L., and the weakness and ataxy of the L. limbs more marked. The deep reflexes present on the R. absent on the L. Plantar reflexes normal. Patient unable to walk alone. A month after this paresis and ataxy of L. limbs was less. She could walk alone, but deviated and stumbled to L. Considerable lordosis in erect attitude. Ocular movements normal.

Six months after operation patient was able to walk quite well, but still tended to stumble to L. Reflexes, normal. The paresis and ataxia of L. limbs had disappeared.

Case 7.—Gumma of the left lateral lobe of the cerebellum.

H. M., aged 27, male. Under the care of Dr. Buzzard, 1904.

Patient had previously malarial fever and syphilis.

For two years attacks of occipital headache accompanied by vomiting and frequently by vertigo, in which he felt as if he himself and the external objects moved from L. to R. Also, noises in his L. ear. A few months later his gait became affected, he staggered and stumbled to the L. It was also noticed that when he stood up there was marked lordosis. Two months before admission all his symptoms became more intense, he had diplopia and failure of vision and occasional attacks of transient blindness. For a month he had lain in a comatose state.

State on Admission.

He was very drowsy and his respiration was periodic.

Special senses.—Smell and taste normal. Hearing, slight deafness on L. side. Vision much impaired; intense optic neuritis.

C. N., 3, 4, 6.—Weakness of the L. external rectus. Slow, deliberate jerking nystagmus on looking to L. Pupils large, equal reacted slowly.

5.—Slight weakness of the L. masseter and also slight L. hypoæsthesia.

7.—Weakness of the R. lower face.

9, 10, 11, 12.—Articulation slow and swallowing difficult, but no paresis of palate or vocal cords.

Attitude.—In bed he lay rather on his L. side with the occiput approximated to the L. shoulder. When he sat up this position of head was still maintained, and he complained of vertigo, subjective rotation of self and objects from L. to R.

M. S.—Owing to syphilitic changes in both elbow-joints he could not extend his fore-arms fully. There was no muscular wasting apart from that due to these changes. The muscles of the R. side were normal in tone, but on the L. definitely hypotonic. The L. limbs were slightly weaker than the R. Co-ordination was slightly impaired in the R. arm and markedly so in the L. limbs. Babinski's cerebellar sign was present in L. There was no spontaneous tremor.

Gait.—Owing to patient's condition it was judged to be inadvisable to get him out of bed.

Sensory system.—No defect.

Reflexes.—Deep; arm-jerks were present on both sides, L. diminished; knee and ankle-jerks were diminished on the R., absent on the L. The superficial reflexes were brisk and equal on two sides, except L. corneal reflex, which was impaired. Plantar responses, flexor. No sphincter trouble.

Cranium.—No deformity, but tenderness on pressure over L. cerebellar region.

Sir Victor Horsley opened the skull in the L. cerebellar region the following day, and three days later he completed the operation. A gummatous mass which occupied the L. lateral lobe especially in its dorsal and outer aspects, where it had infiltrated the dura, was removed.

Immediately after the operation there was skew-deviation of eyes, the L. being directed downwards and inwards, the R. outwards and slightly upwards (plate ii., fig. 4). Slow nystagmus of both eyes in all movements, also spontaneous nystagmus to L.

Tendon-reflexes diminished on R. side, absent L. When examined next day the position of the eyes was unaltered. Upward and outward movement of L. eye limited, inward of R. impaired. Fine, rapid nystagmus on looking to R., while to the L. the nystagmus was more forcible, slow and deliberate. Pupils dilated. Paresis of L. masseter and face. Impairment of hearing on L. Muscle tone of R. limbs subnormal, L. limbs very hypotonic and flail-like when handled. Great weakness and ataxy of L. limb. Tendon-reflexes diminished on R., absent on L. No sensory change.

A month after operation the skew-deviation of the eyes still persisted; nystagmus unaltered. Weakness of L. trigeminus had disappeared and that of L. external rectus and face was less marked. Optic neuritis subsiding and sight improving. Still considerable paresis, hypotonicity and ataxy of L. limbs, while the R. were almost normal. Unable to stand. Tendon reflexes diminished in R. limbs, absent in L.. Plantar flexor responses. Occasionally forced rotation to R. Two months after operation he was much stronger, no headache, but frequently vomited. On pressing on wound vertigo, the subjective rotation of external objects and self being from L. to R. Occasionally forced rotation to R. Vision improved to $\frac{6}{9}$. Optic neuritis completely subsided.

Skew-deviation of eyes had almost disappeared, but still weakness of conjugate movement of eyes to L. Nystagmus slow and coarse to L., fine and rapid to R.

No peculiarity in attitude except slight lordosis and tendency to lean to L. Paresis, hypotonicity and ataxia of L. limbs much less. Could not execute rapidly repeated movements with L. arm. Could just walk alone, inclined trunk to L., and deviated, stumbled and fell to L. Rotation of trunk to R., *i.e.*, L. shoulder forwards.

Tendon-reflexes brisk, L. slightly greater than R. Intermittent, irregular ankle clonus on L. Skin reflexes, normal.

Case 8.—Tumour (glioma) involving the middle lobe and in part the lateral lobes of the cerebellum.

E. B., aged 14, female. Under the care of Dr. Bastian. 1904.

Patient's family history and previous health unimportant.

For eighteen months she had severe pain in the occipital and R. temporal regions, associated with vomiting, retching and nausea. These symptoms were very constant and severe. Eight months ago her eye-sight began to fail, and she suffered a good deal from noises in her head "like bells ringing," but no deafness. Four months later her hands became unsteady, especially the R., and two months later her walking was affected. She staggered like a drunken person, more often to the R. than to the L.

During the last ten months she frequently had attacks of vertigo, generally a feeling as if she and the room were revolving from L. to R., although occasionally it was in the reverse direction.

State on Admission.

Special senses.—Smell and taste normal. Hearing.—Slight deafness L. ear. Vision.—R., $\frac{6}{9}$; L., $\frac{6}{12}$. Intense optic neuritis.

C. N., 3, 4, 6.—Weakness of deviation of eyes to L. Occasionally nystagmus on lateral movement of eyes, especially to L. Pupils equal, reacted normally.

5.—Slight loss of sensation L.

7.—Weakness of L. face, both expressional and volitional.

9, 10, 11, 12.—Functions normal.

Attitude.—Occiput approximated to L. shoulder; R. shoulder held higher and in front of L.

M. S.—No rigidity or flaccidity of limbs. Strength poor on both sides, especially of L. limbs. Inco-ordination of both sides, greater L. than R. Difficulty in rapidly repeating movements with arms, especially L.

Gait.—Stood with body bent over to the L. and feet apart; staggered, reeled and fell to both sides, more frequently to L.

She stands better on the R. leg than on the L.

Sensory S.—Unaffected, save on face.

Reflexes.—Deep; arm jerks equal and brisk, knee and ankle jerks absent. Superficial; present more R. than L. Plantars flexor. No sphincter affection.

Cranium.—No bulging. Tenderness on pressure over occipital region.

During the next month the deep reflexes were at times brisk and equal, but again disappeared, and there was a definite increase in the weakness and inco-ordination of the L. side. The optic neuritis had also increased.

Sir Victor Horsley then removed, in small pieces, a large tumour, which lay in the medial portion of the left lateral lobe, in the inferior portion of the vermis, and extended into the R. lateral lobe at a deeper level than on the L.

The patient recovered well from the operation, but for many days after there was a condition of general asthenia and loss of all deep reflexes, also extreme inco-ordination of all limbs, and inability to sit up or stand.

During the next month the patient's condition improved; there was a steady return of strength, and co-ordination and the deep reflexes reappeared. Two months after the operation the weakness and ataxia were almost limited to the L. side. Slow and deliberate nystagmus on looking to the L., fine and rapid to the R.

The optic neuritis had subsided. The other cranial nerve functions were normal. She could walk with assistance, but tended to stagger and fall to the L., sometimes backwards and to the L.

After five months there was little further improvement. Occasionally headache and frequently vertigo on abrupt movement, the subjective movement of objects and of self being from L. to R., and often forced movements, rolling over to R. side. The nystagmus persisted; L. limbs very flaccid and weaker than R. Power poorly sustained. Ataxy of L. limbs, and some intention-tremor of R. Could only walk with assistance, deviated to L., and fell to either side, most frequently to L. Tendon reflexes brisk. Mental state fair.

Case 9.—*Tumour* (*glioma*) *of the vermis cerebelli.*

C. W., aged 6 years, male. Under the care of Dr. Risien Russell. 1902.

The patient had never been strong and had suffered from rickets. The present illness came on abruptly three weeks before his admission to hospital. He suffered from frontal headache, vomiting, and unsteadiness in walking, his sight became misty, and his mother noticed that his eyes were "queer" and that his hearing was impaired. His condition progressed rapidly and he became drowsy and irritable and frequently soiled himself.

State on Admission.

Very drowsy and sleepy. Severe headache.

Special senses.—Smell and taste, normal. Hearing, impaired on the L.

Vision.—Could only count fingers at ten feet. Severe optic neuritis in both eyes.

C. N., 3, 4, 6.—Weakness of both external recti. Coarse, slow, deliberate nystagmus on lateral deviation to either side, more so to L. than to R. Pupils large and equal, reacted normally to light and convergence.

5, 7, 9, 10, 11, 12.—Functions normal.

Attitude.—When in bed he always lay on his R. side with his head somewhat retracted. When standing there was no peculiarity in the way he held his head.

M. S.—Muscles were well developed, tone was deficient. The strength on both sides was fair and about equal. Co-ordination was impaired on both sides, more so on the L. There was no tremor when at rest.

Gait.—He stood with considerable lordosis, and his feet wide apart. He reeled and staggered to both sides, but more frequently to the L. When his feet were placed together he tended to fall backwards and to the L.

Sensory S.—No defect was noted.

Reflexes.—Deep: increased on both sides, especially on the L. Superficial reflexes present on both sides. The plantar reflexes extensor. No sphincter trouble.

Cranium.—Definite bulging in the L. occipital region with tenderness on pressure. Percussion of the skull yielded a "cracked-pot" note. Considerable hydrocephalus.

Two days after admission the patient became unconscious, his breathing periodic, and the pulse small and rapid. All the reflexes were absent. This condition passed off in about two hours time. On the following day Mr. Ballance opened the skull over the L. cerebellar region. No evidence of tumour in the L. lobe was found, although before the dura was opened there was great intradural tension and a large quantity of cerebro-spinal fluid escaped when it was incised. The after history of this case is of interest, as the patient had a series of fits which were carefully observed. The patient died the day after the operation, and at the autopsy it was seen that there was considerable destruction of the L. lateral lobe of the cerebellum and a hæmorrhage in the region of the nucleus dentatus. A large tumour measuring about 3½ cms. across lay in the central portion of the vermis, extending between the middle cerebellar peduncles, but further to the L. than to the R.

Case 10.—*Tumour (glioma) involving the vermis and both lateral lobes of the cerebellum, chiefly right.*

M. McC., aged 8, female. Under the care of Dr. Risien Russell. 1904.

The family history and previous health of the patient unimportant.

For fifteen months had suffered from constant severe headache and vomiting, the pain often radiating into her neck. This lasted for a month, but recurred again two months later. Shortly after this her eyesight began to fail and her walking became affected; she staggered about like a drunken person. For eight months before admission she was unable to walk, and noticed that the R. hand was unsteady. She never had vertigo or auditory disturbances.

State on Admission.

Special senses.—Smell, taste, and hearing normal. Vision.—R., blind; L., could count fingers. Post-neuritic atrophy of optic discs.

C. N., 3, 4, 6.—Weakness of R. external rectus, and slighter of L. Slow, jerky nystagmus on lateral deviation to the R. Pupils large, the R. inactive to light; the L. reacted sluggishly.

5, 7, 9, 10, 12.—Functions normal.

Attitude.—The occiput held approximated to the R. shoulder, and the chin directed to the L.

M. S.—Muscles were large, and the tone and strength were good on the two sides. Co-ordination was impaired in both R. limbs, and to a lesser degree in the L. upper extremity.

There was no tremor in the extended and unsupported limbs.

Gait.—There was considerable lordosis when erect. The R. leg was abducted and rotated outwards, and she used it awkwardly. The R. shoulder was generally kept in front of the L. She could only walk with help, and tended to fall backwards and to the R. She was able to run fairly well.

Sensory S.—Unaffected.

Reflexes.—Deep ; all increased on both sides. The superficial reflexes diminished on both sides. The plantar reflexes extensor. There was no sphincter trouble.

Cranium.—Considerable hydrocephalus, with bulging of the R. occipital region, and tenderness on pressure over it.

Sir Victor Horsley operated three days after admission. The skull was thinned in the R. occipital region, and a large tumour was found in the R. lateral lobe, and extending into the middle lobe. The greater part of this was removed.

The patient stood the operation well. There was later weakness of deviation of the eyes to the R. with nystagmus. The limbs, especially the R., were very hypotonic and weaker and more ataxic than before operation. The deep reflexes were diminished on the R. Recovery seemed well assured till three weeks later, when she suddenly died.

At the autopsy it was found that only part of the tumour had been removed. A large, spherical mass of soft consistence lay in, and practically destroyed the greater part of the middle lobe, and extended into both lateral lobes, chiefly into the posterior part of the R.

Case 11.—*Tumour* (*cystic glioma*) *of the middle lobe of the cerebellum.*

W. W., aged 26, male. Under the care of Sir William Gowers. 1902.

The family history and previous health of the patient unimportant. Three years previous to his admission to hospital, he had suffered from severe headache and vomiting. He was admitted to a general hospital, and trephined over the L. cerebellar region for the relief of those symptoms. This gave him freedom from the headache for some months, but owing to its recurrence he was again trephined a year later, this time over the R. cerebellar region, but without relief. Two months later the L. lateral lobe of the cerebellum was opened, and a cystic tumour found and removed. After nine months, as the headache had returned, the L. cerebellar region was again explored and some more tissue removed, with, however, only temporary relief. Since then and up to the time of his admission into the National Hospital he had suffered from L. occipital headache, vomiting, staggering gait and attacks of giddiness.

On admission.—Smell, taste, and hearing normal. Vision poor. There was in the eyes a fresh neuritis on the top of a secondary atrophy.

C. N., 3, 4, 6.—No ocular palsy, no nystagmus. Pupils equal and reacted well.

5, 7.—There was slight impairment of the motor functions of these nerves on the R. side.

Attitude.—Owing to constant severe headache when he lay flat, the patient always had his head raised when in bed, and usually lay on his R. side. When standing, the occiput was approximated to his R. shoulder.

M. S.—The tone and development of the muscles were good on both sides. The strength of the two sides was about equal. Co-ordination was impaired on both sides, more on the R. than the L. There was no tremor when the limbs were horizontally extended in front of him.

Gait.—Was reeling and staggering to both sides. Frequently vomiting on movement.

Sensory S.—Unaffected.

Reflexes.—Deep; all increased, R. more than L. Superficial reflexes were normal. Plantars flexor. No sphincter affection.

Cranium.—In L. occipital region there was a trephine opening, the size of a crown-piece, through which a soft pulsating tumour protruded. On the R. side there was a smaller trephine opening, but no bulging. Sir Victor Horsley operated some weeks later, and exposed the dura mater more freely over both sides of the cerebellum, but no tumour was seen. There was considerable hæmorrhage and shock; the dura was incised, and the wound closed. The patient was much collapsed, and died several hours later. The autopsy revealed a large cystic growth, which involved the greater part of the middle lobe and the median portion of the dorsal surface of each lateral lobe, especially of the R.

Case 12.—*Tumour* (*glioma*), *left extra-cerebellar.*

S. R., aged 42 years, female. Under the care of Dr. Beevor. 1903.

Previous health and family history good.

Six months before admission to hospital she began to reel about as if drunk when walking, and her gait rapidly became worse. She also had severe headache, generally occipital, associated with pain and stiffness in neck, and often vomiting. Frequent attacks of giddiness, in which external objects seemed to move in front of her from L. to R., while there was a sense of rotation of self on her vertical axis in the opposite direction. If standing would fall at once, generally forward and to the L. Later the character of the vertigo changed; everything would seem to grow dark, severe tinnitus in L. ear and violent headache set in; then she would vomit; during these attacks would feel either very cold and shiver, or flushed and hot, and sweated profusely. For six months she had complained of deafness and tinnitus in the L. ear. Recently had transient amblyopia.

State on Admission.

Special senses.—Smell and taste normal. Hearing, R. good; L. lost. Sight, could only count fingers at a short distance. Intense optic neuritis.

C. N., 3, 4, 6.—No diplopia or squint, but full lateral deviation of the eyes to the L. was difficult; they could not be long maintained in that position, and quickly receded medialwards. Nystagmus on lateral movements, slower and coarser on looking to the L. than to R.

7.—Slight weakness of L. side of face of peripheral type.

5, 9, 10, 11, 12.—Normal.

Attitude.—R. ear approximated to R. shoulder, but this position not constant. Considerable lumbar lordosis when in the erect position.

M. S.—Muscles well developed, tone and strength good. Well-marked inco-ordination of the L. limbs, chiefly of the arm, and slight unsteadiness of the R. hand in finer movements.

Gait.—Very reeling and unsteady of the drunken type, considerable lordosis; L. shoulder held in front of and higher than R. Constantly deviated and stumbled to the L. Base rather wide. Stands much better on the R. foot alone than on the L.

Sensory S.—No change in any form of sensation detected.

Reflexes.—All normal, and equal on the two sides. Plantars flexor.

Cranium.—No deformity. Some tenderness on pressure in the occipital region.

During the next ten days the patient became much weaker, and on a few occasions fell into a state of coma after severe attacks of headache. Sir Victor Horsley attempted to remove the tumour, but the patient died in a sudden attack of coma four days after the first stage of the operation, *i.e.*, opening of the skull.

At the autopsy a small firm ovoid tumour about three cms. in diameter was found, attached by a pedicle to the membranes under the L. lateral lobe of the cerebellum.

Case 13.—*Tumour* (*fibroma*), *left extra-cerebellar.*

C. H., aged 56 years, female. Sir William Gowers. 1903.

Had good health previous to present illness.

For two years had occipital headache and pain in back of neck, which increased in frequency and severity. Occasional attacks of vertigo, in which she tended to rotate and fall to L.

Also deafness of L. ear with tinnitus. During the three months previous to admission there was often diplopia, her sight had become bad, and she had pain in and weakness of the L. side of her face.

State on Admission.

Special senses.—Smell and taste normal. Hearing, R. fair; L. lost. Sight, could only count fingers at a few feet. Intense optic neuritis.

C. N., 3, 4, 6.—No ocular palsy. Slight weakness of conjugate deviation of eyes to L. Unsteadiness of eyes on looking to R.; slow coarse nystagmus on looking to L.

No other cranial nerve affection.

Attitude.—Nothing noteworthy.

M. S.—Tone and strength of the muscles equal in the limbs of the two sides. A variable amount of tremor in the outstretched arms.

Co-ordination rather poor on both sides, but one arm was not definitely more ataxic than the other.

Gait.—Could walk alone fairly well, but staggered a little; did not deviate or stumble to either side, but seemed more uncertain on the L. than on the R. foot. If an attack of vertigo occurred when she walked, she rotated on her vertical axis to the L., and fell to the L. and backwards. Stood better on R. than on L. foot.

Sensory S.—No change.

Reflexes.—Deep, equal and brisk on the two sides. Superficial, normal. Plantars flexor.

Cranium.—No deformity or tenderness.

An attack of vertigo was observed a few days after her admission to hospital. It began with very severe headache, buzzing in

L. ear, and a feeling of coldness and numbness in the lower extremities. She at once rotated on her vertical axis to the L., and fell to the L. and backwards. Apparently there was no sensation of movement of external objects. Immediately after the attack both arms were very tremulous, there was considerable weakness of the L. side of her face and convergent strabismus, owing to paresis of the L. external rectus. Conjugate deviation of the eyes to the L. was also enfeebled.

Ten days after admission Sir Victor Horsley removed a firm ovoid tumour, measuring 4 by 2½ cms. from the L. ponto-cerebellar region. The patient died two days later from secondary hæmorrhage, but during the time she lived the deep reflexes of the L. limbs were absent, though those of the R. remained brisk.

Case 14.—*Tumour* (*glioma*), *left extra-cerebellar.*

H. C., aged 40 years, male. Under the care of Dr. Tooth. 1903.

Had syphilis 11 years previously. Health otherwise good.

Twelve months before he came under observation he had begun to suffer from severe occipital headache, which persisted. For the same time his L. ear had been becoming deaf, his memory was deteriorating, and he had been irritable and depressed. For six months had frequent attacks of giddiness, in which objects seemed to move in front of him from L. to R., generally no sense of movement of self. If standing, would fall forwards or to L. His gait had been affected for five months. He staggered, reeled, and deviated to the L. For three months had diplopia ; found some difficulty in swallowing, and his speech had been slurred and indistinct.

State on Admission.

Special senses.—Smell normal. Taste good on R., almost absent on L. side of tongue. Hearing good in R. ear, lost in L. to both aerial and bony conduction. Vision natural ; no optic neuritis.

C. N., 3, 4, 6.—Paresis of each external re[illegible]eater L. than R. Weakness of conjugate, lateral moveme[illegible]h sides, but greater to L. On looking to R. fine rapid n[illegible] slower and coarser jerkings on looking to [illegible]impaired, but on looking up there w[illegible] R. to L. Pupils equal and reacted [illegible]

5.—No masseter weak[illegible] on L. side of face.

7.—Slight weakness [illegible]

9, 10, 11 [illegible]

ing solids, and fluids regurgitated through nose. Articulation nasal and slurred.

12.—Tongue protruded straight.

Attitude.—Nothing noteworthy.

M. S.—Muscles well developed, no rigidity or hypotonicity on either side, L. limbs considerably weaker than R. Co-ordination of movements of R. limbs fair, but considerable defect in the L. limbs, greater in arm than leg. Both arms tremulous when horizontally extended in front of him.

Gait.—Very unsteady and reeling, of the drunken type. Could scarcely walk alone. Constantly deviated to the L., saying "something seemed to draw him in that direction"; stumbled and fell to L. Stood more steadily on R. foot than on L. L. leg was held abducted, and rotated outwards.

Sensory S.—No change, save on L. side of face.

Reflexes.—All normal. Plantars flexor.

Cranium.—No bulging or local tenderness.

From the date of his admission into hospital he rapidly became worse, and suddenly died from respiratory failure.

At the autopsy a spherical tumour in part cystic lay on the L. side, between the pons and cerebellum, and excavating both considerably. It measured about 4 cms. in diameter. Microscopically it was a cystic glioma evidently of cerebellar origin, and largely degenerated in parts.

Case 15.—*Tumour* (*glioma*), *left extra-cerebellar.*

F. T., aged 32, male. Under the care of Dr. Ferrier. 1903.

His previous health had been excellent

For nine months he had suffered from severe headache, generally occipital, with pain in neck. Also occasional attacks of vomiting and vertigo, of the latter no definite description was obtained; progressive deafness of L. ear; numbness of L. side of his face and some difficulty in mastication were also of nine months' durat[illegible] For four months there had been some affection of artic[illegible] slight difficulty in swallowing, and he had staggered a[illegible] like a drunken man when walking. For two months[illegible] requ[illegible]opia and sight had failed.

[illegible] *sion.*

S[illegible] te lost on L. side of tongue. [illegible] t. Vision poor, intense very drunken fashion. Great tendency to deviate to the L. of the line of progression, and stumbled more to the L. than R. Could not stand on the L. foot alone.

Sensory S.—No affection of any form of sensation.

Reflexes.—Deep; brisker on R. than L. Abdominal absent on R. Plantar flexor responses on both sides.

Cranium.—Some tenderness in the occipital region.

The patient was operated upon a few days after admission by Sir Victor Horsley, and an irregular-shaped tumour (maximum diameter about 4 cms.) was removed from the L. posterior fossa. He recovered well from the operation, but died a few days later from septic infection, which probably resulted from opening the mastoid cells.

Case 17.—*Tumour* (*glioma*), *left extra-cerebellar.*

R. J., aged 46 years, male. Under the care of Dr. Hughlings Jackson. 1903.

Previous health had been excellent.

For two years he had been aware of gradually increasing deafness of L. ear, and more recently there was constantly a buzzing sound in it. The other symptoms were only of two months' duration. They were: attacks of giddiness in which he became blind and seemed to rotate on his vertical axis from R. to L.; in these attacks has often fallen to the L., unsteadiness in walking apart from giddiness, with a tendency to stumble to L. Intense pain in the occipital region, which radiated down neck. Numbness of L. side of face.

State on Admission.

Special senses.—Smell normal. Taste; good on R., absent on L. side of tongue. Hearing; R. normal, L. diminished, air and bone conduction. Vision good. No optic neuritis.

C. N., 3, 4, 6.—No squint or diplopia. Coarse, slow nystagmus on looking to L. and downwards, fine on looking to R. and up.

5.—Motor functions normal. Considerable anæsthesia on L. side of face.

7.—Paresis of L. side of face of peripheral type.

9, 10, 11, 12.—Palate; L. side weak. Tongue protruded slightly to R. No difficulty in swallowing or affection of speech.

Attitude.—Occiput approximated to L. shoulder, chin directed to R.

M. S.—Muscles well developed. Tone and power good on each side. No inco-ordination. Tremor of both outstretched hands.

Gait.—Walks in a drunken fashion with a constant tendency to deviate to the L. Stumbles and falls to L. No lordosis or other abnormality in attitude.

Sensory S.—No change, save on L. side of face.

Reflexes.—Deep; present and equal on the two sides. Superficial; normal.

Cranium.—No bulging or tenderness.

From the time he entered hospital the patient slowly became worse, and his gait was so much affected that six weeks later he could not walk alone, then slight improvement set in.

State Three Months after Admission.

Severe and persistent occipital headache. Optic neuritis had developed, and the attacks of giddiness were more frequent, in which he felt forced to roll over to the L. in bed, but never noticed movement of external objects. The L. ear had become completely deaf, but the hearing in the R. remained good.

C. N., 3, 4, 6.—Eyes constantly conjugately deviated to R. Weakness of L. external rectus. Nystagmus rapid and fine towards R., slow and coarse to L. Rotatory nystagmus to L. on looking up. No spontaneous nystagmus.

5, 7.—Facial anæsthesia and paresis increased.

9, 10, 11, 12.—Palate weak on L. Difficulty in swallowing and regurgitation of fluids through nose. Speech nasal and indistinct. Tongue protruded to R.

Attitude.—Occiput approximated to L. shoulder. Considerable lordosis when standing.

M. S.—Tone and power of muscles good and equal on the two sides. Great inco-ordination of L. limbs and slight irregularity of finer movements of R.

Gait.—Very ataxic. Could scarcely walk alone. Constantly deviated to L.; remarked spontaneously, "I am always going to the L.; I cannot help it." Stumbles and falls to L. L. shoulder held in front of, and higher than, the R., and L. leg abducted and rotated outwards. Stands more steadily on the R. than on the L. foot.

Sensory S.—Unaffected.

Reflexes.—Normal.

Operation.—A large bilobar tumour, measuring 3½ × 2½ cms., was removed by Sir Victor Horsley from between the L. side of the pons and cerebellum. Immediate recovery from the operation was excellent, but the wound later became septic, and the patient died.

Case 18.—*Tumour, left extra-cerebellar.*

A. W., aged 42 years, male. Under the care of Sir William Gowers. 1904.

Previous to present illness he enjoyed excellent health. Eighteen months ago noticed some deafness in L. ear, which had been complete for nine months. Never tinnitus aurium. For a year daily attacks of vomiting; also headache always worse over the L. eye, but recently also occipital. For ten months very frequent attacks of vertigo; its subjective phenomena were: (1) false projection of objects, they always seemed to the R. and nearer to him than they were; (2) the floor would seem to rise up towards his face, but he could always locate it correctly with his feet; (3) the path on which he walked appeared "corrugated" for the next ten to twenty yards in front of him. Even though its surface was quite level he would for this space see it raised in a series of elevations, more or less regularly arranged, at distances of eight to ten feet from apex to apex. If he stood still there was no movement of the surface of the path, but as he walked "the waves undulated in front of him," keeping a constant distance from him and preserving their relation to one another. This phenomenon would appear and disappear quite suddenly and would recur for shorter or longer periods (from five seconds to five minutes); (4) if walking with a wall on his L. side the wall might appear similarly corrugated in the vertical plane; (5) objects would seem to move in front of him from L. to R. There was no subjective rotation of self, but he would walk in a "stumbling curve" to the L.

For eight months he had occasionally diplopia and his gait was affected. He staggered like a drunken man, but always deviated and stumbled to the L. During the three months before his admission his hands had been unsteady, his sight had failed and he had found some difficulty in swallowing and in articulation.

State on Admission.

Special senses.—Hearing; R. normal; L. absent. Vision, poor, intense optic neuritis.

C. N., 3, 4, 6.—No ocular palsy, but conjugate movement of both eyes to the L., weak and performed only with great effort. Slow, coarse nystagmus on looking to L., irregular and more intermittent to R. Also irregular vertical nystagmus on looking up and down.

5.—Slight anæsthesia on L. side of face and mouth. Masseters equally strong.

7.—Some weakness of L. side of face of peripheral type.

9, 10, 11, 12.—Functions normal.

Attitude.—Occiput always held approximated to L. shoulder. Lordosis as he stood.

M. S.—Development, strength and tone of muscles equally good on the two sides; considerable coarse tremor in both hands when held out in front of him. Some inco-ordination of the L. limbs, and in rapidly-repeated movements the L. arm does not move so freely as, and tires more quickly than, R.

Gait.—Varied very much, independently of vertigo. Sometimes needed assistance. A constant tendency to deviate to the L. and he staggered and fell to the same side. The actual deviation was greater than the apparent as he rotated his trunk on its vertical axis, L. shoulder in front of R., so that he faced to the R. of the line of progression. He recognised the tendency to deviate to the L., and occasionally, when walking fairly well, over-corrected it by this rotation of his trunk; then walked to the R. of the point he wished to reach. His basis was always broadened by abduction and outward rotation of his L. leg. Could stand on the R. foot alone, but not on the L.

Sensory S.—No change in any form of sensation.

Reflexes.—Deep and superficial equal on the two sides. Plantar flexor.

Cranium.—No bulging or tenderness.

Ten days after admission a tumour was removed by Sir Victor Horsley from the L. ponto-cerebellar region. It was spherical in shape and about 4 cms. in diameter, with a cyst placed on its upper surface. In its removal the L. auditory, facial, and trigeminal (sensory portion) nerves were destroyed.

The patient recovered slowly from the operation. After the first two weeks the deep reflexes were exaggerated, but R. more than L., and an extensor response was obtained in the R. foot. There was considerable weakness of the L. limbs, and they were very ataxic; the R. were only slightly unsteady. His state a month after operation was:—

Hearing.—R., normal; L., complete deafness.

C. N.—Great weakness of conjugate deviation of eyes to L., and slow, deliberate nystagmus on attempting to do so. Fine rapid nystagmus to R. Complete anæsthesia and paralysis of L. side of face.

M. S.—Left limbs very flaccid and hypotonic; no difference in strength. L. limbs very ataxic. Occasionally general tremulousness of head and limbs. Could not perform the pronation-supination with L. arm. No rotation movements or vertigo.

Sensory S.—No change.

Reflexes.—Equal and brisk on the two sides. Plantar—R. extensor, L. flexor.

Four months after operation the patient was considerably stronger. Flaccidity of L. limbs still definite, and they were very ataxic, though less so than formerly, but quite as strong as the R.

After six months began to walk alone, but fell, stumbled and deviated to L. Tremulousness disappeared and ataxia was less. No recurrence of vertigo in any form. Nystagmus still persisted.

Case 19.—*Tumour* (*Fibroma*), *left extra-cerebellar.*

W. M., aged 39, male. Under the care of Dr. Risien Russell, 1903.

Previous history unimportant.

For some years his walking had been unsteady and he had always kept some distance apart from his friends when walking, as otherwise he bumped into them. Eighteen months before admission into hospital he began to suffer from headache, vomiting and giddiness. The headache at first was frontal, but later became occipital and bilateral. The vomiting and giddiness were always present when the headache was severe.

During the last twelve months he became deaf in the L. ear, and his vision began to fail. Often diplopia and at times transient blindness. His speech became slow and indistinct.

State on Admission.

Smell normal. Taste defective on R. Hearing defective on L. Vision.—$\frac{6}{8}$ in both eyes, fields contracted. Intense optic neuritis.

C. N., 3, 4, 6.—Pupils equal, reacted normally. Weakness of L. external rectus. Lateral movements impaired to both sides. Nystagmus only occurred on lateral movements; fine and rapid to the R., slow and deliberate to the L.

5.—Slight affection of both motor and sensory divisions on the L.

7.—Slight weakness of the L. lower face.

9, 10, 11, 12.—Tongue flabby and tremulous, and was protruded to the L. Weakness of L. side of palate and of L. sternomastoid and trapezius.

Attitude.—The head was held slightly retracted with the occiput approximated to [illegible] shoulder; there was slight lordosis.

M. S.—Muscles were [illegible] and good tone on both sides, but the power of the L. limbs was considerably less than that of the R. Co-ordination was good on the R., but much impaired on the L. On extending the arms there was a considerable degree of coarse tremor, especially of the L. There were no spontaneous movements.

Gait.—Stood on a wide base with considerable lordosis and intermittent action of the spinal muscles. The L. shoulder was in front of the R. The L. leg abducted and rotated outwards, and its movements ataxic. Often walked towards the R., but invariably staggered and tended to fall to the L. He could stand on the R. leg for a short time, but not on the L. alone.

Sensory S.—No defect except that mentioned above (L. 5th).

Reflexes.—Deep, were all brisk, R. more than L. Epigastric and abdominal present, R. diminished. Plantar reflexes extensor. No sphincter trouble.

Ten days later Sir Victor Horsley removed a hard spherical tumour, measuring about 3½ cm. in diameter, from the L. posterior fossa, lying under the cerebellum near the seventh and eighth nerves on that side.

After the operation there was increase of the weakness of the L. sixth and seventh nerves, and for a time almost complete loss of conjugate movement of the eyes to L. Fine rapid nystagmus on looking to R., slower and coarser on attempting deviation to L. The patient was for some time quite blind. Articulation was slurred.

The power of the two sides was about equal, but the L. limbs were hypotonic and very ataxic. The tendon reflexes, which were lost after operation, returned in about two weeks and were eventually exaggerated, especially on L. side. Plantar reflexes flexor.

Patient continued to improve for two weeks, then became very irritable and mentally affected. This phase lasted about a month, then steady improvement set in and he became bright and intelligent.

Three months after operation the weakness of lateral movement of eyes to L. and the nystagmus were still present. Tone and power of limbs were good. Great ataxia of L. limbs, though movements of the R. were well co-ordinated. He could not execute rapidly-repeated movements with L. arm. He could walk with slight assistance. Sensation was unaffected, and the tendon-reflexes were brisk and equal on the two sides.

Seven months after operati[illegible]ould walk alone with very little unsteadiness, but still s[illegible]cy to deviate and stumble to L. His vision had impr[illegible]arse nystagmus on looking to L. but no other disturb[illegible] nerve functions.

Case 20.—*Tumour, right extra-cerebellar.*

C. T., aged 32 years, male. Under the care of Dr. Beevor: 1904.

Previous health and family history excellent.

For 16 months indefinite affection of gait. All the other symptoms were of 6 months' duration. Then began to walk like a drunken man, and became unable to turn quickly. Had frequently attacks of giddiness of which no definite description could be obtained, "things seemed to be mixed up anyhow," but there was never any sensation of movement of self. The vertigo as a rule only came on when he moved quickly, and the apparent movement of external objects was in the opposite direction to that in which he moved his head. In the attacks felt inclined to fall, generally backwards. For six months, too, the R. ear had been deaf; he had frequently diplopia and occasionally difficulty in swallowing. Had only had one short attack of headache and vomiting.

State on Admission.

Special senses.—Smell and taste good. Hearing, R. ear deaf to both aerial and bony conduction; L. natural. Vision poor; intense optic neuritis.

C. N., 3, 4, 6.—Slight weakness of R. external rectus. Slow coarse nystagmus was occasionally visible on looking to R.

5.—No affection of motor or sensory functions.

7.—Slight paresis in expressional and volitional movement of R. side.

9, 10, 11, 12.—Weakness of R. side of palate. Tongue protruded straight.

Speech, nasal. Slight difficulty in swallowing solids.

Attitude.—Nothing noteworthy.

M. S.—No difference between the tone of the muscles of the two sides, but the L. limbs were considerably weaker than the R. Co-ordination impaired on both sides, more so in L. than in R., and there was some intention-tremor on finer movements; also some coarse tremor in the horizontally-extended arms.

Gait.—Like that of a semi-drunken person, staggered, especially on turning. Constantly deviated to the R., but generally stumbled and fell to the L. Feet kept rather wide apart.

Sensory S.—Unaffected.

Reflexes.—Deep; all exaggerated on L., normal on R. Plantar extensor responses.

Cranium.—No local tenderness.

While in hospital his gait became more unsteady, the optic neuritis increased and his vision deteriorated.

State Five Weeks after he came under Observation.

Hearing.—R. quite lost; no tinnitus. L. good.

C. N., 3, 4, 6.—No ocular palsy observed. Nystagmus often accompanied lateral movement, but was not constant or regular. It most frequently resulted on looking to R., and was then always slow and forcible, being finer, rapid and intermittent on lateral movement to L. No nystagmus on vertical movement.

5.—No sensory loss on face, masseters equally strong.

7.—R. facial weakness, more marked in expressional than in volitional movement.

9, 10, 11, 12.—R. side of palate weak. Articulation nasal. Stridor during sleep, but vocal cords moved well. Tongue straight.

Attitude.—Occiput frequently approximated to R. shoulder. No lordosis.

M. S.—Tone of muscles about equal on the two sides. Some weakness of L. limbs resembling that of spastic hemiplegia, though the R. were not so strong as they had been. Tremor in the outstretched hands. Inco-ordination greater in R. than L. limbs, Babinski's pronation-supination test less well carried out L. than R.

Gait.—Walked on a broad basis with R. leg somewhat abducted. R. shoulder generally held in front of and higher than L. Deviated to R. but stumbled and fell to L. With feet together fell to either side.

Reflexes.—Deep; exaggerated on L., normal on R. Abdominal diminished on L. plantar; R. indefinite response, L. extensor.

Owing to the increase of the symptoms an attempt was made by Sir Victor Horsley to remove the tumour, but the patient died the evening after the first stage, *i.e.*, removal of the bone, was completed.

At the autopsy a large tumour, measuring 5½ × 4 × 3½ cms., was found lying between the pons and the under surface of the R. lateral lobe of the cerebellum. It excavated both, but did not directly involve either.

Case 21.—*Tumour* (*fibroma*), *left extra-cerebellar.*

J. J., aged 30; male. Under the care of Sir William Gowers. 1903.

The only noteworthy fact in the previous health of this case was a doubtful history of syphilis.

Three months before admission into hospital he had suffered from severe headache, chiefly frontal; also diplopia, and for the last two weeks had been unable to walk, and became very drowsy, with occasional incontinence of urine.

State on Admission.

On admission he was dull and irritable.

Special senses.—Smell and taste were unimpaired. Hearing was diminished on the L. Vision—R. $\frac{6}{24}$; L. $\frac{6}{18}$; no optic neuritis.

C. N., 3, 4, 6.—Weakness of the L. external rectus, no nystagmus. Pupils equal and reacted well.

5.—Slight motor weakness of the L. trigeminus.

Other cranial nerves were normal.

Attitude.—He showed tendency to hold his head retracted.

M. S.—Power was good on both sides. No rigidity or flaccidity. Co-ordination was good.

Gait.—Uncertain, with a constant tendency to fall backwards.

Sensory S.—Was unaffected.

Reflexes.—Deep; all increased, R. more than L. Plantar reflex on R. side extensor; on L. flexor. Slight incontinence of urine.

His condition gradually became worse, and a month later was as follows:—

Well-marked optic neuritis; L. ear deaf.

C. N., 3, 4, 6.—Weakness of L. external rectus; nystagmus on looking to L.

5.—Motor and sensory impairment of L. side very marked, with loss of taste on L. side of tongue.

7, 9, 12.—All weak on the R. side.

M. S.—Very little loss of power, but marked ataxia of both upper limbs, and to a less degree of the lower limbs, the L. being more affected than the R. There was no spontaneous tremor.

Reflexes.—Deep: all much increased, with ankle clonus on both sides, the increase being more on the R. than L. Plantar: R. extensor, L. indefinite. Incontinence continued.

Sensory.—No change except on face.

Cranium.—No bulging, tenderness in occipital regions on both sides.

Sir Victor Horsley removed a large growth from the L. posterior fossa a few days later. The patient recovered from the operation and improved steadily for over a fortnight; then he became dull and irritable, and refused his food. His temperature rose; he developed pneumonia and died semi-delirious.

At the autopsy it was found that a considerable portion of the tumour had not been removed; the remains lay in the L. surface of pons excavating the ventral surface of cerebellum. Its greatest bulk lay at level of fifth nerve.

Case 22.—Tumours (fibromata), extra-cerebellar, in each side.

R. S., aged 16 years, male. Under the care of Dr. Taylor. 1903.

Always delicate. Cataract in R. eye at five years of age.

For two years had tinnitus in both ears, and gradually became deaf. Completely deaf in both ears for one year. Two years previously also began to stagger, he could not walk straight, gait gradually became worse, stumbled like a drunken man, but not more to one side than to the other. For three months the sight of his L. eye had been deteriorating, and he had had several transient attacks of blindness. For two months occasional headache, but no vomiting. No vertigo.

State on Admission.

Special senses.—Smell and taste normal. Hearing, deaf in each ear to both aerial and bone conduction. Sight—R. nil, opaque cataract; L. poor, intense optic neuritis.

C. N., 3, 4, 6.—Weakness of each external rectus, greater on L. than on R. Vertical movements and convergence unaffected. Nystagmus on lateral movement, more forcible in looking to L. than to R.

5, 7, 9, 10, 11, 12.—Functions normal, except that the L. side of the palate was weak, and the articulation slurred.

Attitude.—Head generally bent forwards. Trunk also bent forwards when standing, and stood on a broad basis like an old man.

M.S.—Tone and strength equally good on each side. Limbs rather unsteady when held out. Slight ataxia of both upper extremities, generally greater in L. than R. Rapidly-repeated movements fairly well done on either side.

Gait.—Walks very unsteadily, like a drunken man, with feet far apart. No marked tendency to deviate to either side, bu stumbles to both, chiefly to L.

Sensory S.—No affection.

Reflexes.—Equal and brisk on the two sides. Plantar responses flexor.

Cranium.—Some bulging of L. occipital region. No tenderness.

During the next two months the patient became weaker, and his gait much more ataxic. Sometimes the inco-ordination was more marked in the L., sometimes in the R. limbs.

He was then operated upon by Sir Victor Horsley, and an irregular ovoid tumour about 4 cms. in diameter was removed from the L. posterior fossa; considerable injury to that lobe of the cerebellum resulting, as the growth was taken out through it. For some hours after the operation well marked "skew-deviation" of the eyes, the L. being directed down and inwards, the R. slightly up and outwards.

The patient recovered fairly rapidly from the operation.

The deep reflexes were for a time diminished in the L. limbs, but soon became brisk and equal. For some weeks there was considerable weakness and flaccidity of the L. limbs, and they were very ataxic in their movements.

State Six Weeks after Operation.

Hearing.—Quite deaf in each ear. Sight $\frac{6}{36}$, optic neuritis subsided into atrophy.

C. N.—Weakness of L. external rectus, coarse nystagmus on looking to R. and L., more regular and deliberate in latter direction.

Considerable paresis of L. side of face, of peripheral type.

Articulation nasal and indistinct, but no paresis of palate.

Attitude.—Generally bent forwards. Occiput approximated to L. shoulder.

M. S.—Muscles hypotonic, especially those of L. limbs, which are much weaker than R. Ataxia of limbs of two sides, much greater L. than R. Hands very tremulous when held out in front of him.

Gait.—Could only walk a few steps alone, deviated to L., and reeled and stumbled in same direction. Fell always to L.

Sensory S.—No change detected.

Reflexes.—Equal and normal in type on the two sides.

The patient's condition remained more or less unchanged while he was under observation. After several months he became very drowsy and lost the power of walking. He died at home eleven months after the operation.

Autopsy.—The greater part of the L. lateral lobe of the cerebellum was destroyed, and a collection of cerebro-spinal fluid separated its remains from the dura mater. A large spherical firm tumour lay in the R. posterior fossa compressing the pons and cerebellum on that side. It was loosely adherent to the membranes.

Reviews.

Les Psychonévroses et leur traitement moral. (Leçons faites à l'Universite de Berne.) Par le Dr. DUBOIS, Professeur de Neuropathologie. Préface du Dr. DEJERINE). Paris, Masson et Cie., 1904. Pp. 557.

THIS is a book of considerable interest. It is obviously written by a physician of experience and judgment, who has thought out for himself the problems presented to him by his patients. The advice given is, speaking generally, excellent and marked by common sense. Unfortunately the author has not learned the ar of condensation, and the book consequently suffers from being too long. It contains a number of accounts of cases, given in a conversational manner, with reports of the colloquies that took place between the doctor and patient. The reports are well told and often witty, and, from the author's point of view, illustrate his method of treatment, in which advice and moral direction play the principal part, but, on the other hand, they are somewhat vague as regards clinical signs and symptoms, and the reader has often to take the diagnosis for granted. Essentially the book is a development and expansion of the theme that the most important part of the treatment of the various neuroses lies in the moral influence of the physician over the patient, and that success in treatment is to be attained, with certain minor adjuvants, by exerting this moral force or domination. This is, of course, by no means a new discovery, and the author is far from claiming it as such, but he thinks that up to the present time its value has not been fully utilised, although one would have thought that the influence of the mind on the body has been pretty much in the air of late, and that the physician's attention is too frequently drawn away from this, which he regards as the only permanent method of cure, to the passing fashion in treatment, whatever it be—hypnotism, electric baths, the high-frequency current, or radiotherapy. No one would dispute the original proposition that by force of character or will-power, to use a popular term, the physician may often, if he can get the patient placed under favourable conditions, achieve wonders in the way of cure of the neuroses. Indeed, regarded from this point of view, a large part of all medical treatment is a kind of suggestion. We must, however, be careful in talking of suggestion, for the author prefers the word persuasion or moral suasion to describe his method, avoiding the word suggestion because it is mostly used in a bad sense. He quotes von Bechterew's saying that suggestion comes in by the back stairs, whilst persuasion enters at the front door. He, quite rightly, protests against the employment of hypnotic suggestion, as not justifiable in the interests of the patients, often, indeed, constituting a kind of moral vivisection. He would reserve hypnotic suggestion for exceptional use with due precaution in certain obstinate cases of hysteria, and generally substitute for it moral suasion. With moral suasion he combines where necessary other modes of treatment, and seems mostly to employ in suitably-selected cases the Weir-Mitchell cure, with its main factors of isolation, rest, over-feeding, and massage, varying the use of each of these means according to individual needs. As showing a minor point of difference between the two countries, little or no stress is laid upon the importance of ensuring a suitable nurse for the cure, indeed, judging from one or two hints, and also from the statement that the conversational powers of the masseuse afford a pleasant relief to the monotony of isolation, the inference is that a special nurse is at any rate not always employed.

There is, of course, no doubt of the influence that a doctor of forceful character may obtain over his patient, and especially over a hysterical or neurasthenic one; but, unfortunately, this is just one of the qualities of a doctor that "is born and not made," and cannot be imparted by much taking of thought. At the same time a book of this kind may be read with profit, especially by a man just beginning practice; for although, in the absence of natural aptitude, it cannot give him the art, it can be of great help in aiding him to learn how to advise and to treat his neurotic patients.

There is another aspect of the moral influence of the doctor over his patients, of which readers of Oliver Wendell Holmes' "Elsie Venner" will not need to be reminded, and that is, that in patients of the opposite sex this influence, good in its first effects, may ultimately become prejudicial to their welfare, and lead to unforeseen difficulties.

To give a further account of the book, it opens with the discussion of the psychological problems which underlie such a system of psychotherapeutics. We soon find ourselves, like our first parents in the Garden—and we may say, in passing, that the first recorded instance of suggestion occurred there—engaged in a discussion on free-will and determinism, with a passing glance at predestination. It is needless to remark that the conclusions arrived at are not those of Milton. The chief symptoms of neurasthenia next receive consideration, the leading characteristic being held to be readiness to experience fatigue (*fatigabilité*). The author deems it unnecessary for his purpose to describe the symptoms of hysteria, which he regards, with certain exceptions, as *une maladie psychique par excellence*, as *ideogenic*. He would class both diseases together as different manifestations of a general state of nervousness (nervosisme). It is difficult, however, to see the advantage of so doing.. No doubt the present views of these disorders will undergo considerable modification in the future, but advance rather lies in the contrary direction of further and more exact differentiation.

The author makes some sound observations on the necessity of treating the general state in the neuroses; too often in such cases the patient goes from one specialist to another, according as the symptoms vary from time to time. "One is neurasthenic," according to him, "just as one is lazy, tired, or irritable." The chapter on sexual disorders conveys sensible advice, with a caution against a too elaborate investigation of the patient's sexual history. The value, at times, in both hysteria and neurasthenia, of a studied neglect of symptoms in promoting their disappearance, whilst at the same time the patients are unconsciously helped on to a better state of mind, is insisted upon in several places.

In the last chapter Dr. Dubois gives a summary of his position. For the prevention of the neuroses he looks with greatest hope to the cultivation of the intellectual and moral faculties, rather than to the development of the body. The present rage for athletics finds no support from him; he remarks that the latter-day equivalent for *mens sana in corpore sano* seems to be that the cultivation of the body necessarily *entails* a sound mind. In this age of hurry he insists on the necessity for a due amount of repose. The maxims of Seneca are several times quoted and recommended to patients for their inward digestion; and the author's ideal of life seems to be a genial stoicism to carry through the difficulties of the hour.

In conclusion, the author writes well, and although one may not agree with all his views, and may possibly think that the leading idea is sometimes pushed somewhat far, yet his advice is marked by original thought, sound sense, and is evidently based on a considerable experience. There is, however, much repetition, and the book would gain in value by a certain amount of condensation.

J. MICHELL CLARKE.

Organic Nervous Diseases. BY M. ALLEN STARR, M.D., LL.D. Baillière, Tindall & Cox, London, 1903.

THIS is a book of great merit. It combines in its 700 pages a synopsis of current knowledge with the results of years of observation by a most able and experienced clinician. For this reason the book is more than a text-book, and may be regarded as a valuable contribution to neurological literature. Perhaps for the same reason it is slightly unequal in accordance with the interest taken by the author in one or other subject. Nothing, for instance, could be better than the chapters dealing with diseases of the peripheral nerves, with diagnosis and location of diseases of the spinal cord or of the brain, and they stand in marked contrast with the description of the symptomatology of tumours of the cerebellum. It would need considerable good fortune for anyone to make a correct diagnosis of the presence of a cerebellar tumour, or to state whether it was situated in the right or left, or middle lobe, from the information supplied in the page and a half devoted to the subject. The absence of any mention of hemiparesis, of inco-ordination apart from that suggested by the word "staggering," or of the diagnostic value of head positions, are noteworthy.

An Englishman will hardly be likely to agree with the statement that multiple sclerosis is a very rare affection, but th author is not alone among his countrymen in affirming the infrequency of the disease in America. At the same time, it is rather a matter of regret, from the students' point of view, that the cases cited in this connection are so atypical, and have not been the subject of pathological verification. In spite of a few minor defects, the book is one which may be confidently recommended to neurologists, not only on account of its originality and the general excellence of the descriptions, but also for the valuable hints on treatment which it provides and for the beautiful pathological and clinical illustrations, which are more numerous and better reproduced than in any text-book on the subject with which we are acquainted.

E. FARQUHAR BUZZARD.

Contributions from the William Pepper Laboratory of Clinical Medicine. (University of Pennsylvania. No. 4, 1903, Philadelphia.)

THIS volume contains twenty-nine articles which are either reprints from papers already published or read before various societies. Sixteen of them deal with the nervous system, and of these four are by Dr. William G. Spiller, who also participates in seven others. It is not possible to notice all the valuable papers collected in this volume.

The first paper is by Dr. Spiller, and describes the results of unilateral degeneration of the glossopharyngeal and vagus nerves in a case of fracture of skull.

A man, aged 54, fell and fractured his left occipital bone into the foramen magnum—bony fragments pressing upon the nerves of the medulla. The patient lived for forty-six days. The essential points in the clinical history were complete paralysis of the left half of the larynx, and complete inability to swallow. As the patient declined to be fed by the nasal tube, rectal alimentation had to be employed.

At the autopsy an area of hæmorrhage and softening was found on the under surface of the cerebellum. The roots of the left glossopharyngeal and vagus nerves were intensely degenerated. A point of great interest was that the sterno-mastoid and trapezius muscles were not paralysed, showing that the association of the spinal accessory nerve with the lower vagus roots within the jugular foramen cannot be very intimate.

The left lung was congested and contained numerous areas of consolidation, some breaking down into abscesses the size of a split pea. The right lung showed a similar condition confined to the middle lobe. This bronchopneumonic condition is not uncommonly observed after injury of the vagus. From the fact that the taste buds had disappeared from the circumvallate papillæ on the left side of the tongue, the author concludes that they must be innervated by the glossopharyngeal nerve, and he quotes in support a case of Rosenberg's in which a similar degeneration occurred in a man in whom the ninth nerve had been destroyed by a tumour. Observations on the condition of the pulse were too scanty to be of any use.

Dr. Spiller also gives an account of the pathological changes in the nervous system in a case of lead poisoning.

The patient was a man, aged 48, who had worked in lead for twenty years. He had had attacks of lead colic; a characteristic blue line was present on the gums; slight wrist drop was present with complete paralysis of legs, incontinence of urine and delirium. While in hospital the paralysis of the lower limbs largely disappeared, while that in the upper extremities became more marked. No mention is made of alcohol as a possible additional factor.

The case, therefore, was one eminently suitable for examination. The anterior cornual cells of the cervical and lumbar regions were intensely degenerated; the lumbar posterior roots were little altered. The median nerve showed marked degeneration by Marchi and Weigert hæmatoxylin stain, and the extensor muscles of the forearm showed much degeneration of nerve bundles within the muscles. In the brain, the chief change noticed was proliferation of the endothelial cells on the outer surface of the pia.

The other papers are mainly reports of interesting cases, with one experimental research on regeneration of posterior spinal roots. Among the papers not relating to the nervous system, two are of especial interest, one a careful report of a case of chronic acetanilid poisoning with marked alterations in the blood, by Alfred Stengel and C. Y. White; the other, a discussion on methods of determining blood pressure in man, by W. B. Stanton.

There are numerous illustrations throughout the volume, and altogether it does great credit to the laboratory from which it emanates.

A. E. Russell.

Vorlesungen über den Bau der Nervösen Zentralorgane des Menschen und der Thiere. By Dr. Ludwig Edinger. Erster Band, "Das Zentralnervensystems des Menschen und der Säugethiere." Seventh Edition, Leipzig, 1904.

The seventh edition of Dr. Edinger's well-known work is to be published now in two volumes. The first volume here reviewed deals with the central nervous organs of men and of mammals, and the author has made many alterations rendering this volume still more valuable than its predecessors to the physician and the pathologist, which embody the results not only of pure anatomical research, but also of pathological and experimental investigation.

In order to facilitate topographical comprehension of transverse sections of the nervous system, the pictures of the transverse section appear as the anterior or posterior surface of the nervous system cut transversely and drawn in relief. The relation of internal structure to the external conformity, always a matter of difficulty to the student, is clear from the first glance at these admirable pictures. The number of these illustrations in relief is not so extensive as the student of serial sections might wish, but each subdivision of the brain stem and spinal cord is figured.

The paucity or absence of explanatory lettering to some of the illustrations both upon the plates and in the text, while it adds to the clearness and artistic effect, necessitates a careful reading of the context.

The first four chapters deal with the methods of investigating the anatomy of the nervous system, and with the general structure, physiology and development of nervous organs. An historical survey of the chief workers in this branch of science from the time of Vesalius to the present day is given, and the methods of research used by these pioneers is briefly described. In the description of the neuroglia, the author inclines strongly to the opinion that in diseases of the nervous system glial proliferation is always secondary to degeneration of the nerve elements. The present state of our knowledge with regard to the neuron hypothesis is clearly laid out. Great importance is attached to the existence in post-embryonic life of incompletely developed nervous paths. The attainment of physical and mental culture by practice is accompanied by the opening up of such paths and the complete medullation of their fibres. The capacity for anatomical development in the central nervous system persists throughout life as long as the capacity for learning remains.

The special anatomy of the mammalian central nervous organs is described in the form of nineteen lectures, the human brain being taken as the type. The comparative development of the human brain is first dealt with, and the relations of its parts to those of the brains of lower animals is made beautifully clear by the superposition of the outlines of the mammalian brain in red upon a black relief drawing of a Teleostean brain.

The macroscopic anatomy of the brain and spinal cord is most lucidly described, the omission and inclusion of details being selective with regard to their physiological and pathological utility.

The description of the segmental innervation of the skin and of the muscles has been previously published by Dr. Edinger. It is much at variance both with the opinions of the highest authorities, and with clinical facts. It has been put forward as tentative only, but even at that it is not in keeping with the high standard of excellence of the book. The region of the umbilicus is certainly supplied from the ninth and not from the tenth dorsal roots. Such areas as are represented for the segmental skin supply of the legs are never seen clinically. On the other hand, the prolongations upwards of the L 3, L 4, S 1, S 2 areas, are probably the correct solutions of a much-debated question. The meeting of the area C 2 with the area of the fifth nerve in front of the angle of the jaw as figured, must surely be a mistake in the lettering, for it is contradicted by the area marked C 2 upon the occipital region.

The structure of the spinal cord is described with great clearness, and without any superfluous detail. Dr. Edinger's description of the ending of the pyramidal tracts in the spinal cord leaves the student erroneously convinced, and the critic entirely unconvinced. He states that the fibres of the pyramidal tracts end in the region of the ventral horn cells. Those of the ventral pyramidal tract can readily be traced into the ventral horns, where they can be seen breaking up. But the collaterals from the lateral pyramidal tracts cannot be traced into the ventral horns by the Marchi method in the human adult, but can only be followed thither in the embryo, and by the "silver" method. No mention is made of the conspicuous ending of the lateral pyramidal collaterals around the cells of Clarke's column. To argue a similar ending for the ventral and lateral pyramidal fibres, when the former can always be demonstrated and the latter never, is far from conclusive, while to ignore the accumulation of evidence of the highest authorities, that the lateral pyramidal collaterals can only be traced to the region of Clarke's column, is not justifiable.

Fig. 71, page 129, is a diagram representing the various tracts which may present in a transverse section of the cord. Taking a pre-pyramidal position in the lateral column, a large area is marked "tractus thalamo-spinalis." So conspicuous is this large tract upon a most important diagram that it is most disappointing to find no reference to it in the text. The opinion that the loss of pain sense, and of temperature sense, is to be connected with disease of the gray substance of the posterior horns is of great interest.

In the description of the medulla oblongata and pons, Dr. Edinger introduces some innovations of terminology which are most useful. The large system of descending fibres which occupies the middle third of the mesial fillet, and lies between the cortico-bulbar tract (mesial) and the chief sensory path (lateral), and which passes with the fillet fibres through the interolivary layer and ends in the lower medulla, is named the "tractus thalamo-bulbaris"; while the fibres which reach the ventro-lateral region of the cord constitute a "tractus thalamo-spinalis." The so-called juxta-restiform body is named "tractus nucleo-cerebellaris." Fibres passing directly to the vermis cerebelli from the vestibular nerve and from the fifth nerve constitute the "direct sensory cerebellar roots" of these nerves. A tract, the fibres of which arise throughout the sensory nucleus of the fifth nerve, and passing directly across the middle line, collect upon the opposite side in a position lying between the descending root of the fifth and the central tegmental tract to ascend to the thalamus, is named the "tractus quinto-thalamicus."

Of especial interest in the description is the nervous mechanism subserving taste. The terminal nucleus of taste is a mass of gray matter which lies mesial and somewhat dorsal to the restiform body in close connection with the fasciculus solitarius of which it forms the end nucleus. Reaching from the upper cervical cord to the middle of the pons, this nucleus receives fibres from the back of the tongue, &c., *via* the glossopharyngeal nerve and petrous ganglion, and from the front of the tongue (1) *via* the chorda tympani geniculate ganglion and portio intermedia, and (2) *via* the lingual and Gasserian ganglion. This seems to be a happy solution of a much disputed question.

Hellweg's triangle is described as an ascending tract under the name of tract spino-olivaris. The cells of Deiter's nucleus are in close synaptic connection with collaterals of the vestibular nerve, and from these cells arise two most important sets of fibres: (1) the vestibulo-spinal tract; (2) fibres which, crossing to the dorsal longitudinal bundle of the same side and of the opposite side, divide into two, the upper prolongation passing *via* the dorsal longitudinal bundle to the motor nucleus, and the lower passing in the dorsal longitudinal bundle to the ventral column of the cord. This description and the accompanying diagram, fig. 108, will serve to illustrate the clearness and brevity with which the author impresses matters of great fundamental importance upon the student.

The mesial fillet is definitely divided into three groups of fibres—a lateral group, the chief sensory path; a central group, the thalamo-bulbar tract (descending system of fillet); and a mesial group consisting of the pyramidal projection for the motor cranial nerve nuclei.

After stating that the pyramidal projection for the motor cranial nerve nuclei descends in the two positions: (1) by the accessory fillet, and (2) by the mesial fibres of the ventral pyramid, Dr. Edinger inclines to Dejerine's view, that Spitzka's bundle may be a third path for these fibres, and is possibly identical with the accessory fillet, the bundle being sometimes placed dorsolaterally—Schlesinger's accessory fillet, and sometimes mesioventrally—Spitzka's bundle. This opinion does not appear justifiable in the light of the work of Schlesinger, Hoche and Barnes.

The morphological description of the cerebellum is modified according to the recent work of Elliott Smith.

In the diagram representing the connections of the superior peduncle, the lateral bundle of Pròbst, which passes the red nucleus to end in the ventral nucleus of the thalamus, is figured.

The statement that " the symptoms which result from disease of the lateral lobes of the cerebellum are still quite unknown," will not appeal to those who are accustomed to diagnose with accuracy lesions of the lateral regions of the cerebellum by means of the signs which Risien Russell has laid down as symptomatic of such lesions.

To facilitate the comprehension of the symptoms resulting from lesions of the brain stem and thalamus, several illustrations are provided of transverse sections of these regions upon which supposed lesions in various positions are depicted as shaded circles, and the symptoms which resulted from a lesion in each position in actual cases is appended.

The central auditory path is figured as decussating completely in the corpus trapezoides, a semi-decussation of auditory fibres does not occur, and the central acoustic path seems to be in connection with the opposite ear only. The clinical fact, that lesions destroying the lateral fillet or the posterior colliculus do not cause complete deafness of the opposite ear, has therefore no anatomical explanation at present. The descriptions of the oculo-motor mechanism, and of the complex of the dorsal longitudinal bundle, are of particular excellence.

It is of special interest that degenerate fibres can never be traced into the oculo-motor nuclei after destruction of those cortical centres from which ocular movements are readily elicited by stimulation.

The morphology of the cerebral hemisphere is admirably described, Elliott Smith's new terminology being adopted.

The genetically older part of the pallium, the archipallium, consists of a basal portion (olfactory lobe) and a marginal portion (hippocampal formation). The remaining portions of the hemispheres which are of later origin constitute the neopallium, and these first make their appearance between the basal and marginal portions of the archipallium. The anterior commissure is the chief commissure between the lateral halves of the archipallium, the psalterium being a second archipallial commissure. With the development of the neopallium and its large commissure (the corpus callosum), the marginal portion of the archipallium and its commissures (the hippocampal formation and the psalterium) are pressed backwards, and come to occupy a ventro-caudal position, the limbic fissure marking the limit between the archipallium and the neopallium.

The volume closes with an excellent résumé of the subject matter of the second part. The present edition of the work embodies the results of the most recent researches, and it is entitled to be regarded, even more than its predecessors, as a text-book for students quite unrivalled in any language. The text is a model of clearness and of classic German. The illustrations are abundant and beautifully reproduced.

The prominence given both in illustrations and in the text to the works of recent English authors, and especially to those of Dr. Elliott Smith, will be highly gratifying to English readers.

JAMES COLLIER.

L'Epilessia. By P. PINI. (Milan, Ulrico Hoepli, 1904.)

THIS small volume is a résumé of recent work on the subject of epilepsy, and, while not adding anything original, is certainly a full representation of the present state of our knowledge, especially of treatment.

The plan of the book is to discuss first the various theories as to the causation of epilepsy, to examine fully the "fluids," urine, sweat, blood, cerebro-spinal fluid and the gastric juice. According to the finding in each case, so are epileptics to be treated. Thus, diminution of the alkalinity of the blood calls for treatment that will raise the reaction to standard. The next theory discussed is the bacteriological, and the author suggests that at some future time epileptic seizures may be amenable to "serum" treatment. In support of this, he mentions cases which were free from attacks during the course of erysipelas. The greater portion of the book is occupied by a dissertation on the various methods of treatment by different forms of bromides, biborate, hypnotism and baths. Lastly, an account is given of epileptic colonies. Our opinion of the book is that it will prove a useful monograph for the author's countrymen. We cannot say it supplies a want in international medical literature. DOUGLAS STANLEY.

La Nevrasthenia. By Dr. LUIGI CAPPELLETTI. (Milan, Ulrico Hoepli, 1904.)

THE prevalence of neurasthenia in modern life is ample apology for the appearance of any fairly good work on the subject. We believe that Dr. Cappelletti's book is a very complete description of the leading characteristics of the disease, and quite equal to the more usually read accounts of neurasthenia in English. The order followed is convenient, the author giving a general description of the symptoms and etiology, next a minute account of the special senses, general sensation, the respiratory, circulatory, and other systems. The importance of recognising the underlying condition of neurasthenia in some forms of cardiac and gastro-intestinal diseases is of course great, but we notice a tendency on the part of some writers on this subject to attribute to neurasthenia many forms of gastric disease, forgetting that such disease may have induced a general neurasthenic condition. Thus, defects of the pylorus and "coprostasis" are more likely to cause than to be caused by neurasthenia. The present work is open to this criticism. The writer devotes a chapter to the consideration of the leading theories of neurasthenia, such as Bouchard's auto-intoxication, Beard's nervous theory, ending with a criticism of them all.

By far the most thorough part of the book is that dealing with treatment; here the more practical methods of dealing with the neurasthenic state are detailed, such as massage, electricity, baths, Weir-Mitchell's method, and others. In conclusion, we are of opinion that, as a monograph, Cappelletti's work is useful and comprehensive, and a valuable addition to Italian medical literature.

DOUGLAS STANLEY.

Annali della Clinica delle Malattie Mentali e Nervose della R. Università di Palermo. Vol. ii. (Palermo, Fratelli Marsala-Editori, Angolo Via Parlamento.)

THIS collection of monographs appearing together is a variable record of researches carried on at Palermo University for two years. The volume contains a variety of papers, among which must be mentioned "A Study of Tubercular Neuritis." The article is by Colella, and shows evidence of considerable labour. We require further evidence of the occurrence of neuritis in tuberculosis, as in many cases it has not been proved that alcoholism has been altogether excluded. Some of the cases quoted by the writer appear genuine, if we may rely on the history, but, as is well known, patients' statements require to be corroborated. Accompanying the paper are several very carefully executed plates, showing segmentation of the white sheaths, pigmentary infiltration of the cornual cells, vacuolation of nuclei, and the usual lesions found in neuritis occurring under other circumstances. The same writer contributes a paper on "Traumatic Psycho-neuroses," which is interesting and instructive. Another paper of value is "An Investigation into the Effects of Electricity on the Cerebral Pulse," by Emanuele Gentile, in which the writer describes the effect on the amplitude and duration of pulsation in the cerebral vessels when stimulated by faradic and galvanic currents, the latter in different directions. A carefully illustrated paper on the structure of nerve cells is contributed by Messina-Vitrano, based on the methods of Donagio, Cajal and Golgi. Such a collection of papers is a valuable addition to any reference library.

DOUGLAS STANLEY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

A PATHOLOGICAL MEETING was held on Thursday, December 1, 1904, at 8.30 p.m., at 11 Chandos Street, Cavendish Square.

A discussion on "Lesions of the Posterior Columns of the Spinal Cord in Diseases other than Tabes Dorsalis" was opened by Dr. David Orr.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

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EDITOR.

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FOR

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AND

LIST OF OFFICE BEARERS
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OF THE

Neurological Society of the United Kingdom,

1904,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1903.

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Neurological Society of the United Kingdom.

REPORT OF THE COUNCIL FOR 1903.

FEBRUARY 4, 1904.

The Council has much pleasure in reporting the continued increase in the prosperity of the Society, which now numbers 246 members.

It regrets the loss of its honorary member, Mr. HERBERT SPENCER, who died on December 9, 1903, and of its corresponding member, GEHEIMRATH PROFESSOR JOLLY, who died on January 6, 1904.

The Presidential Address given on January 22 by Professor J. N. LANGLEY, F.R.S., on "The Autonomic Nervous System," was fully reported in the Spring number of "Brain." The abstract of the Proceedings of the Society shows the scope and character of its work during the past year. A new feature will be found in the dedication of a whole meeting of the Society to a single subject in Neurology, illustrated by typical and by unusual cases. The meeting devoted to Myopathy in its various forms was a great and encouraging success.

In consequence of the untiring energy and hospitality of the Edinburgh members, the meeting of the Society in that city will remain a most pleasant memory to all who were fortunate enough to be present.

On November 5 the third Hughlings Jackson Lecture was delivered by Sir WILLIAM BROADBENT, Bt., F.R.S.

The outstanding accounts for printing, publishing, and illustrating "Brain" have been a severe strain on the limited funds of the Society, but, in consequence of the care of the Editor and the persistent efforts of the Treasurer, the Society is now again entirely free from debt. Moreover, in addition to a small balance, the remaining copies of the Index to "Brain" are a valuable asset.

It is hoped that the prosperity of the Society will continue to increase, and so enable it to maintain the present high reputation of its Journal "Brain."

Proceedings of the Society during the Year 1903.

January 22.—Dr. J. N. Langley, Inaugural Address, "The autonomic nervous system."

February 26.—Clinical Meeting; Subject, "Myopathy." This meeting was devoted to the exhibition of cases of idiopathic muscular atrophy.

April 2.—Dr. Farquhar Buzzard, "The pathology and bacteriology of Landry's paralysis."

June 27.—Meeting of the Society at Edinburgh.

October 22.—Clinical meeting. Dr. Harris and Mr. Low, "Case of Erb-Duchenne paralysis treated by operation." Dr. T. Grainger Stewart, "A case of diplegia and myoclonus." Dr. Leonard Williams, "A case of infantile hemiplegia of the right side, with paralysis agitans beginning on the left side." Sir Victor Horsley, "Three cases of cerebellar tumour after operation, with reference to the question of cerebellar compensation." Dr. Dundas Grant, "Three cases of nervous lesion, with manifestations in the throat and ear." Dr. Gordon Holmes, "Three cases of unilateral tremor in cerebral lesions." Dr. Collier, "A case of (?) disseminated sclerosis." Dr. F. E. Batten, "A case of (?)." Dr. Farquhar Buzzard, "Progressive muscular atrophy in a young woman."

November 5.—Hughlings Jackson Lecture, delivered by Sir William Broadbent, "Hughlings Jackson as pioneer in nervous physiology and pathology."

December 3.—Dr. Farquhar Buzzard, (1) "Descending spinal fibres of a lumbar posterior root"; (2) "A dermoid tumour of the brain." Dr. Michell Clarke, (1) "Diphtheritic paralysis"; (2) "Gumma of the spinal cord." Dr. H. A. Caley, "Hydatid of the brain." Dr. J. S. Collier, (1) "Fibres of the pyramidal tract ending in Clarke's column"; (2) "Ending of fibres of the sacral triangle"; (3) "Specimens from cases of cerebral diplegia." Dr. W. Harris, "Prefixed and postfixed types of brachial plexus." Dr. James Flashman, "Advantages of the clove oil celloidin embedding method." Dr. Fletcher and Dr. Batten, "The pathology of a case of progressive muscular atrophy in a child aged 11."

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Guinea, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, the Editor of "Brain," and twelve Councillors, of whom not less than three shall be extra-Metropolitan Members.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years, one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him; and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN [illegible] Journal of Neurology," the organ of the Society, and edited for it by R. [illegible] ith, M.D., F.R.C.P.

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1892	H. Charlton Bastian, M.D., F.R.S.
1893	Edward A. Schäfer, F.R.S.
1894	David Ferrier, M.D., F.R.S.
1895	Sir William H. Broadbent, Bart., M.D., F.R.S.
1896	Alexander Hill, M.A., M.D.
1897	George H. Savage, M.D.
1898	Sir Victor Horsley, M.B., F.R.C.S., F.R.S.
1899	W. H. Gaskell, M.D., F.R.S.
1900	Augustus Waller, M.D., F.R.S.
1901	W. Julius Mickle, M.D.
1902	Herbert William Page, M.A., F.R.C.S.
1903	J. N. Langley, D.Sc., F.R.S.
1904	S. J. Sharkey, M.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897	J. Hughlings Jackson, M.D., F[illegible]
1900	Geheimrath Professor Hitzig, [illegible]
1903	Sir William H. Broadbent, [illegible]D., F.R.S.

Elected

1898 Haslett, John Hanfield, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.D., D.Sc., F.R.S.E., University College of South Wales, Cardiff.

1891 Head, Henry, M.D., F.R.C.P., F.R.S., 143, Harley Street, W. (C., 1900; Sec., 1901-1904.)

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.-P., 1895; C., 1892-94.)

1903 Holmes, Gordon M., M.B., B.Ch., National Hospital, Queen Square, W.C.

O.M. Horsley, Sir Victor, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.-P., 1896-97; C., 1892-98, 1899.)

1903 Howland, Goldwin, W., M.B., M.R.C.P., National Hospital.

1889 Huggard, William R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 Hulme, George Frederick, M.B., C.M., South View, Montague Road, Felixstowe.

1901

1899 Fawcett, John, M.D., F.R.C.P., 26, St. Thomas Street, S.E.

O.M. Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1886; V.-P., 1892-3; C., 1886, 1890-95.)

1903 Flashman, J. F., M.B., Ch.M., B.Sc., Totnes, Temple Street, Petersham, Sydney, Australia.

1903 Fleming, Robert, M.D., F.R.C.P.Ed., 10, Chester Street, Edinburgh.

1903 Fletcher, H. Morley, M.D., F.R.C.P., 98, Harley Street, W.

O.M. Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.-P., 1897-98; C., 1896, 1900.)

1903 Gibson, G. A., M.D., D.Sc., F.R.C.P.Ed., 3, Drumsheugh Gardens, Edinburgh.

O.M. Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1902 Goldschmidt, Oscar Bernard, M.B., Bishopstone House, Bedford.

1899 Goodall, Edwin, M.D., F.R.C.P., Joint Counties Asylum, Carmarthen.

1898 Goodliffe, Jno. Henry, M.D.Aberd., 802, High Road, Lower Tottenham.

1897 Gossage, Alfred Milne, M.B., F.R.C.P., 54, Upper Berkeley Street, W.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, J. Dundas, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 Guthrie, Leonard, M.D., F.R.C.P., 15, Upper Berkeley Street, W. (C., 1904.)

1902 Hall, W. Hamilton, M.R.C.S., Fordcombe, Tunbridge Wells.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., West Ham Borough Asylum, Goodmayes, Ilford.

1897 Harris, Wilfred J., M.D., M.R.C.P., 37, Queen Anne Street, W.

Elected

1901 Cox, Joshua John, M.D., St. Ronan's, Clarendon Road, Eccles, Manchester.

1901 Craig, Maurice, M.D., M.R.C.P., Bethlem Royal Hospital, S.E.

1898 Darley-Hartley, W., M.D., M.R.C.P., Main Road, Sea Point, Cape Town, South Africa.

1895 Davidson, Andrew, M.D., Callan Park, Sydney, N.S.W.

1897 Dawson, William R., M.D., F.R.C.P.I., Farnham House, Finglas, co. Dublin, Ireland.

1898 Dodd, Henry Work, F.R.C.S., 186, Harley Street, W.

O.M. Dreschfeld, Julius, M.D., F.R.C.P., 3, St. Peter's Square, Manchester. (V.-P., 1898-99.)

O.M. Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1903 Dudgeon, L. S., M.R.C.P., 6, Powis Gardens, London, W.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

Green, F. W., M.D., F.R.C.S., 14, Welbeck Stree

1889 Humphry, Laurence, M.D., M.R.C.P., Lensfield, Cambridge.

1903 Hunter, Walter K., M.D., D.Sc., 7, Woodside Place, Glasgow.

1901 Hunter, William, M.B., C.M., Government Bacteriological Laboratory, Hong Kong.

1903 Hunter, William, M.D., F.R.C.P., 103, Harley Street, W.

O.M. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.-P., 1887-88; C., 1886, 1890.)

1889 Hyslop, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. Jackson, J. Hughlings, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.). Hon. Memb., 1904.

O.M. Jessop, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 Johnston, George, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1903 Jones, A. E., M.B., B.Sc., University College Hospital, Gower Street.

1892 Jones, Henry Lewis, M.D., F.R.C.P., 143, Harley Street, W.

1894 Jones, Robert, M.D., F.R.C.S, Claybury Asylum, Woodford, Essex.

1902 Kelynack, T. N., M.D., M.R.C.P., 120, Harley Street, W.

1894 Kidd, Percy, M.D., F.R.C.P., 60, Brook Street, W. (C., 1904.)

1896 Langdon, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 Langdon-Down, Reginald L., M.B., M.R.C.P., 47, Welbeck Street, W.

O.M. Langley, J. N., M.A., D.Sc., F.R.S., Hedgerley Lodge, Madingley Road, Cambridge. (Pres., 1903; V.-P., 1901-1902; C., 1898-99, 1904.)

1888 Lawford, J. B., M.D., F.R.C.S., 99, Harley Street, W. (C., 1902-3.)

O.M. Lees, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1901-1903.)

1897 Lewis, H. Wolseley, M.R.C.S., L.R.C.P., London County Asylum, Banstead, Surrey.

1904 Lindsay, J. A., M.D., F.R.C.P., 13, College Square E., Belfast.

1901 Little, E. Graham, M.D., M.R.C.P., 61, Wimpole Street, W.

Elected
1897 Loewenthal, Max, M.D., M.R.C.P., 126, Princes' Road, Liverpool.
1899 Lord, John R., M.B., C.M., London County Asylum, Bexley, Kent.

1898 McAldowie, Alexander M., M.D., 6, Brook Street, Stoke-on-Trent.
1898 MacCormac, John M'Gee, M.D., 31, Victoria Place, Belfast.
1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.
1902 McDougall, Wm., M.B., Weald Mount, Haslemere.
1902 Mackenzie, James, M.D., Bank Parade, Burnley.
O.M. Mackenzie, Sir Stephen, M.D., F.R.C.P., 31, Cavendish Square, W. (C., 1896-98.)
1896 Mackintosh, Ashley W., M.A., M.D., 9, Bon Accord Square, Aberdeen.
1903 Macnamara, E. D., M.B., B.C., 45, Campden House Road, W.
O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)
O.M. Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.
1902 Mantle, Alfred, M.D., M.R.C.P., Savile Place, Halifax.
1901 Marris, W. A., M.D., Springfield Road, King's Heath, Birmingham.
1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.
1891 May, W. Page, M.D., F.R.C.P., Helouan, Cairo, Egypt.
1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.
O.M. Mercier, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1902.)
1897 Meyer, Adolf, M.D., LL.D., Pathological Institute, New York State Hospital, Ward's Island, N.Y., U.S.A.
O.M. Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.-P., 1900; C., 1895-97, 1902-4.)
1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.
O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.
1902 Morrison, C. S., L.R.C.P.E., County and City Asylum, Hereford.
1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1902.)
1901 Mould, George Wm., M.R.C.S., Cheadle Royal Asylum, Manchester.
1899 Mould, Gilbert E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.
1894 Murray, George R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.
1889 Murray, H. Montague, M.D., F.R.C.P., 25, Manchester Square, W.

1904 Neild, Newman, M.D., Richmond Hill, Clifton, Bristol.
O.M. Nettleship, Edward, F.R.C.S., Shottermill, Haslemere, Surrey.
1902 Newington, Hayes, F.R.C.P.Ed., The Gables, Ticehurst, Sussex.
O.M. Nicolson, David, C.B., M.D., M.R.C.P.Ed. Balgownie, Guildford, Surrey.
1888 Niermeyer, J. H. H., M.D., Amsterdam.
1904 Nolan, M. J., L.R.C.P., L.R.C.S.I., Down District Asylum, Downpatrick.
1888 Norman, Conolly, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

Elected

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 Oliver, Thomas, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. Orange, Wm., C.B., M.D., F.R.C.P., Oakhurst, Godalming, Surrey.

O.M. Ormerod, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

1899 Orr, David, M.D., County Asylum, Prestwich, Manchester.

O.M. Page, Herbert William, M.A., F.R.C.S., 146, Harley Street, W. (Pres., 1902; V.-P., 1900-1; C., 1891-92, 1903-04.)

1903 Palmer, Fredk. S., M.D., M.R.C.P., 7, Gordon Place, Gordon Square, London.

1895 Parkinson, John Porter, M.D., M.R.C.P., 57, Wimpole Street, W.

1901 Parsons, John Herbert, M.B., B.Sc., 28, Great Ormond Street, W.C.

1901 Passmore, E. S., M.D., M.R.C.P., Croydon Asylum, Warlingham, Surrey.

1896 Paterson, Donald Rose, M.D., M.R.C.P., 15, St. Andrew's Crescent, Cardiff.

1899 Piper, Francis P., M.B., M.R.C.S., L.R.C.P., King's Leigh, Tankerton, Whitstable.

1888 Pitt, G. N., M.D., F.R.C.P., 15, Portland Place, W. (C., 1903-04).

O.M. Purves, W. Laidlaw, M.D., 20, Stratford Place, W.

1899 Rambaut, Daniel F., M.D., B.Ch., County Asylum, Bicton Heath, Shrewsbury.

1894 Ransom, William B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 Rayner, Henry, M.D., M.R.C.P.Ed., 16. Queen Anne Street, W.

O.M. Reid, E. W., M.B., F.R.S., University College, Dundee.

1897 Reissmann, Charles H., M.B., B.Sc., St. Peter's, Adelaide, South Australia.

1893 Rennie, George E., M.D., M.R.C.P., College Street, Sydney.

1895 Reynolds, Ernest S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1891 Rivers, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1898 Robertson, William Ford, M.D., 7, Hill Square, Edinburgh.

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1901 Rows, R. G., M.D., County Lunatic Asylum, Lancaster.

1892 Ruffer, Marc Armand, M.D., Ramleh, Egypt.

1903 Russell, A. E., M.D., M.R.C.P., 9, Wimpole Street, W.

1889 Russell, J. S. Risien, M.D., F.R.C.P., 44, Wimpole Street, W. (Sec., 1896-1900; C., 1901-1904.)

1899 Russell, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

1901 Salaman, Redcliffe N., M.A., M.B., Frognal End, Frognal Gardens, Hampstead.

O.M. Sanderson, Sir J. Burdon, Bart., M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

O.M. Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.-P., 1895; C., 1886-90, 1898.)

Elected

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.-P., 1892; C., 1886-93.)

1901 SCHORSTEIN, GUSTAVE, M.D., F.R.C.P., 11, Portland Place, W.

O.M. SEMON, Sir FELIX, C.V.O., M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 SEWARD, WM. JOSEPH, M.B., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (Pres., 1904; V.-P., 1902-3.; C., 1899-92.)

O.M. SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.

1898 SIMPSON, FRANCIS O., L.R.C.P., M.R.C.S., Rainhill Asylum, Liverpool.

1900 SINGER, H. D., M.D., M.R.C.P., St. Thomas's Hospital, S.E.

1895 SMITH, F. J., M.D., F.R.C.P., 138, Harley Street, W.

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901-1904.)

1895 SPICER, W. T. HOLMES. M.B., F.R.C.S., 5, Wimpole Street, W.

1901 STAFFORD, WILLIAM, M.B., 110, Mansfield Road, Nottingham.

1895 STANLEY, DOUGLAS, M.D., M.R.C.P., 21, Broad Street, Birmingham.

1895 STANSFIELD, THOS. E. K., M.B., London County Asylum, Bexley, Kent.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., F.R.S., 8, Park Square West, Regent's Park, N.W. (C., 1904.)

1901 STEELL, GRAHAM, M.D., F.R.C.P., 96, Mosley Street, Manchester.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 7, Harley Street, W.

1903 STEWART, T. GRAINGER, M.B., B.Ch., M.R.C.P., National Hospital, Queen Square, W.C.

1897 STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1902 STONE, W. G., M.D., 93, Denmark Hill, S.E.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

1902 SULLIVAN, WM. CHARLES, M.D., H.M. Prison, Pentonville, N.

1904 SUTHERLAND, GEO. ALEXANDER, M.D., F.R.C.P., 73, Wimpole Street, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 TAYLOR, F. R. P., M.D., East Sussex Asylum, Hellingly, Sussex.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W. (C., 1900-3.)

1898 THOMSON, H. CAMPBELL, M.D., F.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., Oxford Lodge, Victoria Park, Manchester. (C., 1901-1903.)

1892 TITCHENER, E. B., B.A., Ph.D., Cornell Heights, Ithaca, N.Y.

O.M. TOOTH, H. H., C.M.G., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 TREVELYAN, E. F., M.D., B.Sc., F.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., F.R.S.Ed., 20, Charlotte Sq., Edinburgh. (V.P. 1903-04.)

Elected

1891 **Tuke, Thomas Seymour**, M.B., Chiswick House, Chiswick.

1891 **Turner, William Aldren**, M.D., F.R.C.P., 18, Harley Street.

1898 **Turney, Horace G.**, M.D., F.R.C.P., 68, Portland Place, W.

O.M. **Tweedy, John**, F.R.C.S., 100, Harley Street, W.

1901 **Vincent, Swale**, M.B., Physiological Laboratory, University College, Cardiff.

1888 **Voorthus, J. A.**, M.D., Apeldoorn, 542, Loulaan, Netherlands.

O.M. **Waller, Augustus**, M.D., F.R.S., 32, Grove End Road, N.W. (Pres. 1900; V.-P., 1899; C., 1894-97, 1901.)

O.M. **Warner, Francis**, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1902 **Warrington, W. B.**, M.D., M.R.C.P., 69, Rodney Street, Liverpool.

1904 **Watson, Chalmers**, M.B., F.R.C.P.E., 22, Coates Crescent, Edinburgh.

O.M. **Watteville, A. de**, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1902; Sec., 1886-89; Editor of "Brain," 1880-1900.)

1897 **Weber, F. Parkes**, M.D., F.R.C.P., 19, Harley Street, W.

1897 **West, Samuel**, M.D., F.R.C.P., 15, Wimpole Street, W.

1897 **White, Ernest W.**, M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. **White, W. Hale**, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1902.)

1894 **Whiting, Arthur J.**, M.D., M.R.C.P., 142, Harley Street, W.

1889 **Wigglesworth, Joseph**, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1900 **Williams, Leonard**, M.D., M.R.C.P., 8, York Street, Portman Square, W.

1894 **Williamson, Richard T.**, M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1896 **Wilson, Albert**, M.D., 1, Belsize Park, N.W.

1903 **Wilson, G. R.**, M.D., M.R.C.P.Ed., 8, Rutland Square, Edinburgh.

1896 **Wood, Guy M.**, M.B., 6, Woburn Square, W.C.

1889 **Wood, T. Outterson**, M.D., F.R.C.P.Ed., 40, Margaret Street, Cavendish Square, W.

1892 **Woodhead, G. Sims**, M.D., F.R.C.P.Ed., 6, Scroope Terrace, Cambridge.

1899 **Wright, Hamilton**, M.D., Pathological Institute, Selangor, Straits Settlement.

1898 **Young, Robert Arthur**, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.